ANNALS OF THE ROYAL COLLEGE OF SURGEONS OF ENGLAND

VOLUME 28 JANUARY 1961 No. 1

CELBENIN

REGD. TRADE MARK BRL 1241

Sodium 6—(2,6—dimethoxybenzamido) penicillanate monohydrate

"A new penicillin has been prepared which is active against the usual penicillin-sensitive microorganisms and yet resists staphylococcal penicillinase. This is a major event in chemotherapy. From the information given in the British Medical Journal last week and in the three papers appearing in our present issue there is good reason to hope that the new BRL 1241 ('Celbenin', Beecham Research Laboratories) will be a means of controlling the staphylococcal infections which have plagued hospitals throughout the world during the past ten years."

EDITORIAL (1960) LANCET, II, 585

Seven papers reporting work on Celbenin appeared in the British Medical Journal of September 3rd, 1960, on pages 687, 690, 694, 700, 703, 706 and 708; three in the Lancet of September 10th, 1960, on pages 564, 568 and 569.

BRL

CELBENIN is a product of British Research at
BEECHAM RESEARCH Laboratories Ltd. Brentford England

TRAINING MADE EASIER FOR THE COLOSTOMY PATIENT

Training is the key to normal living for the colostomy patient. ★SPENCER'S new colostomy support — *individually designed, cut, and made* for each patient— makes training easier, because:

★ Abdominal section opens instantly by means of zippers—facilitating the change of pads

★ This section is lined with moisture-proof material—easily cleaned—protects outer clothing

★ Spencer's co-relation of abdominal and back support improves posture, body mechanics

★ Cosmetic results are appreciated by both male and female patients

★ *The Care of the Permanent Colostomy, Can. Med. Assoc. Jr. 60 : 71-72 (Jan.) 1949.*

For further information write to:

—— SPENCER (BANBURY) LTD. ——

Consulting Manufacturers of

Surgical and Orthopaedic Supports

Spencer House, Banbury, Oxon.

Tel. 2265

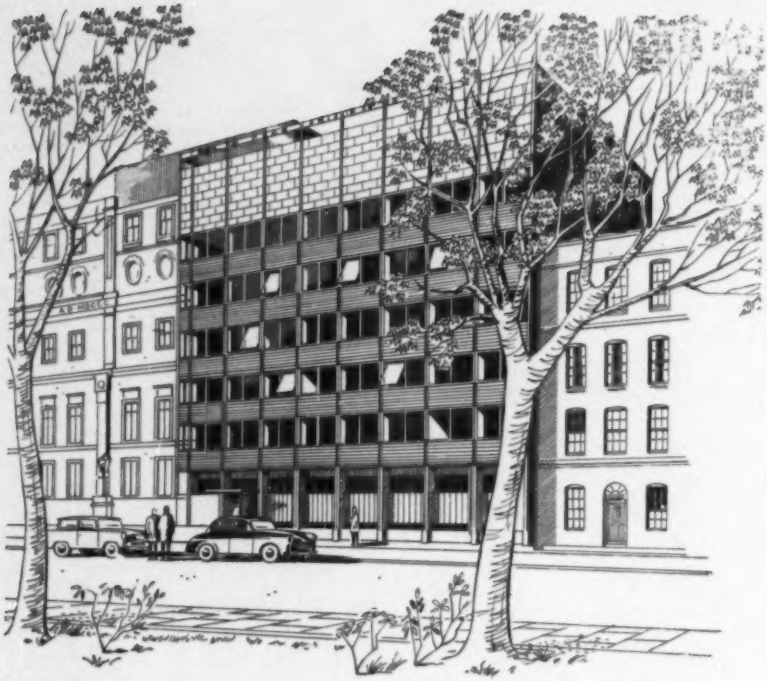
Branch Offices:

LONDON: 2, South Audley Street, W.1 ... ... Tel. GROsvenor 4292
LIVERPOOL: 79, Church Street, 1 ... ... Tel. ROYal 4021
LEEDS: Victoria Buildings, Park Cross Street, 1 (opposite Town Hall Steps) Tel. Leeds 3–3082
BRISTOL: 18, Whiteladies Road (opposite Broadcasting House), Bristol, 8. Tel. Bristol 36410
MANCHESTER: Room 9, 2nd Floor, Mitton Hall, 244 Deansgate, Manchester 3 Tel. Blackfriars 7822
GLASGOW: 86, St. Vincent Street, C.2 ... ... Tel. CENtral 3232
EDINBURGH: 30a, George Street, 2 ... ... Tel. CALedonian 6162

Appliances supplied under the National Health Service through Subsidiary Company Spencer (Surgical Supplies) Ltd.

Trained Spencer Retailer-Fitters resident throughout the Kingdom. Name and address of nearest Fitter supplied on request.

Copyright



Help build this great Cancer Research Centre

The fight against cancer—mankind's cruellest enemy—is being intensified. The Imperial Cancer Research Fund seeks your help in building new laboratories in Lincoln's Inn Fields. Due to open next year, this will be the most advanced research centre in Europe. But £1,000,000 is needed to complete it.

The Imperial Cancer Research Fund, pioneer organisation of its kind, was founded by the Royal Colleges of Physicians and Surgeons to undertake this vital work in its own Laboratories.

To widen its activities and to speed results, the Fund—which receives no official grant—makes this *personal* appeal to you. Will you please help—*now*?

IMPERIAL CANCER RESEARCH FUND

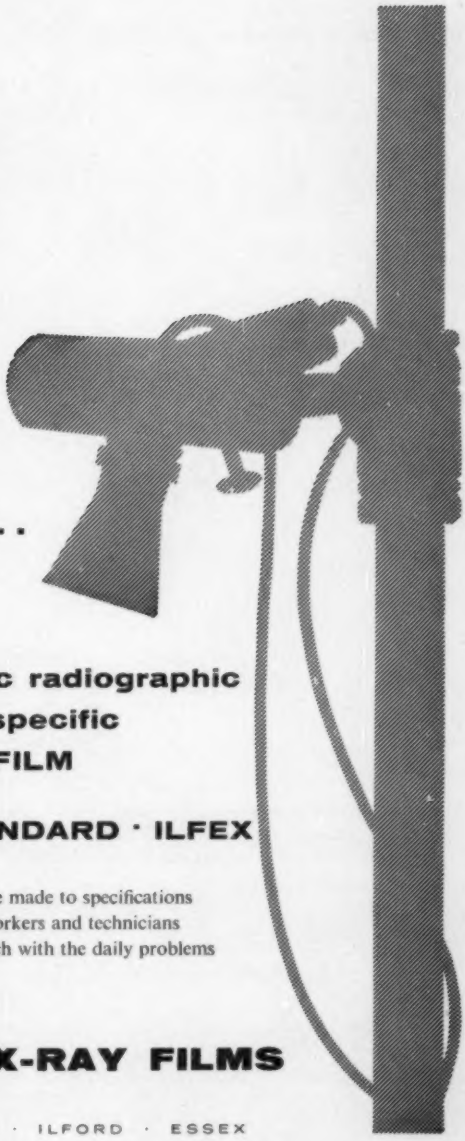
PATRON: HER MAJESTY THE QUEEN

Please send your gift to **A. Dickson-Wright Esq.,** M.S., F.R.C.S., **C.R.F. 94, Imperial Cancer Research Fund, 49 Lincoln's Inn Fields, London, W.C.2**

Diagnosis

Routine

Research . . .



for every specific radiographic need there is a specific ILFORD X-RAY FILM

RED SEAL · STANDARD · ILFEX

ILFORD X-ray films are made to specifications drawn up by research workers and technicians who are in constant touch with the daily problems in radiography.

ILFORD X-RAY FILMS

ILFORD LIMITED · ILFORD · ESSEX

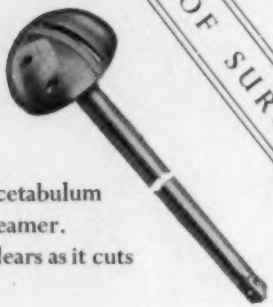
PRYOR & HOWARD LTD.

Office and Works: WILLOW LANE, MITCHAM, SURREY

TELEPHONE NO.: MITCHAM 1177-8

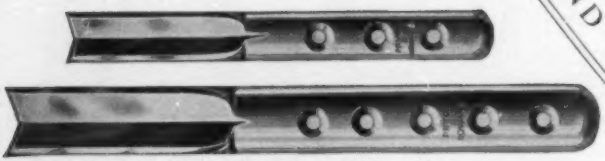
MAKERS OF SURGICAL INSTRUMENTS AND APPLIANCES

Modified
Jewett Brace
with
self-aligning plates



Acetabulum
Reamer.
Clears as it cuts

Polythene and
Plastic
appliances



L. C. Wood V-Blade Plate

Write for Leaflets and Catalogues

LONDON FITTING ROOMS: **49, WELBECK STREET, W.1.** *Telephone No.: WEL 2782*

BRANCHES: **LEEDS · BOURNEMOUTH · BRISTOL**

'Fluothane'

for Obstetric

Anaesthesia

* *Induction is rapid and smooth and is sufficient to allow application of forceps.*
* *There is a striking absence of reflex movement with manipulation under very light anaesthesia.*
* *Laryngospasm does not occur and there is no stimulation of salivary or bronchial secretions.*
* *Little tendency to vomiting and retching.*
* *Condition of the infant is satisfactory.*
* *Post-partum uterine atony and haemorrhage unlikely.*
* *Recovery is rapid without complications.*

'Fluothane' has been widely used in obstetric anaesthesia, for caesarian section, forceps delivery and for normal confinements. It presents useful advantages over other anaesthetic agents and has been described as the anaesthetic of choice for obstetrics.*

* *Canad. Anaes. Soc. J.* (1960), **7**, 109

Fluothane – an I.C.I. discovery

(Halothane B.P.C.) Trade Mark

Literature and further information available on request.

ICI

FH80

IMPERIAL CHEMICAL INDUSTRIES LIMITED PHARMACEUTICALS DIVISION WILMSLOW CHESHIRE

A *new* Colour Vision Test

by Professor KAKICHI UMAZUME of Tokyo Medical College

PRINCIPAL FEATURES

1. Each colour chip painted by hand from media prepared to Spectrophotometric data and of enduring qualities superior to printers' colours.
2. Accuracy of screening. Differentiation between protans and deutans to an accuracy of 99·1%.
3. Simplicity of classification including plates for determining degree of abnormality.
4. Definite grading excludes malingering.
5. Inclusion of new plates for screening tritanopes.

Technical Pamphlet on application.

Price £7 7 0

CURRY & PAXTON LTD.

195-199, GREAT PORTLAND STREET *and* 22, WIGMORE STREET, LONDON, W.1
INSTRUMENT DEPT: **120 Albert Street, London, N.W.1.** *Tel.:* GUL 7939
Branches throughout the country.

ROSE & CARLESS
Manual of Surgery

19th Edition

Consulting Editor: SIR CECIL WAKELEY, Bt., K.B.E., C.B., LL.D., M.Ch., D.Sc., F.R.C.S., F.R.S.E., F.A.C.S., F.R.A.C.S.
Joint Editors: MICHAEL HARMER, M.A., M.B., B.Chir.(Cantab), F.R.C.S., and SELWYN TAYLOR, M.A., D.M., M.Ch.(Oxon), F.R.C.S.
Rose & Carless is known to generations of students as a singularly comprehensive textbook of general surgery. The new edition is virtually a new book almost complete rewriting and the addition of some 370 new illustrations made necessary by recent advances in surgery and related disciplines. The revision includes new chapters on malignant disease, the endocrine system and paediatrics; and orthopaedic surgery, surgery of the nervous system and of the alimentary tract, the management of electrolyte balance and chemotherapy have received particular attention.

"The whole text is lucid and well written. The illustrations are outstanding. There can be little doubt that **Rose & Carless** *will still retain its place as the best textbook of surgery for students and practitioners."*
BRITISH JOURNAL OF SURGERY

19th Ed. 1404 pages 940 illus. 12 coloured plates 84s. postage 2s. 9d.

BAILLIÈRE, TINDALL & COX

7 & 8 Henrietta Street · London · WC2

Polybactrin

antibiotic powder spray

This new presentation of POLYBACTRIN is the result of intensive research into the mechanics of pressure packing antibiotics in powder form. While the formula and potency of POLYBACTRIN remain unchanged, radical modification of the valve mechanism and design of the unit provides several advantages :— **positive directional, wide-angle spray gives wider coverage of the area under treatment — all metal container provides additional safety — special valve eliminates impacting of powder and clogging of nozzle.**

The use of Polybactrin during surgical procedures is a positive method of inhibiting wound pathogens which may become implanted in the tissues.

INDICATIONS : General surgery — gynaecological operations — orthopaedic surgery — Burns etc.

FORMULA : Powder content 1.5 g.
Each gramme contains :

Neomycin Sulphate (as base)	330 mg.
Polymyxin B Sulphate	100,000 units
Zinc Bacitracin	25,000 units

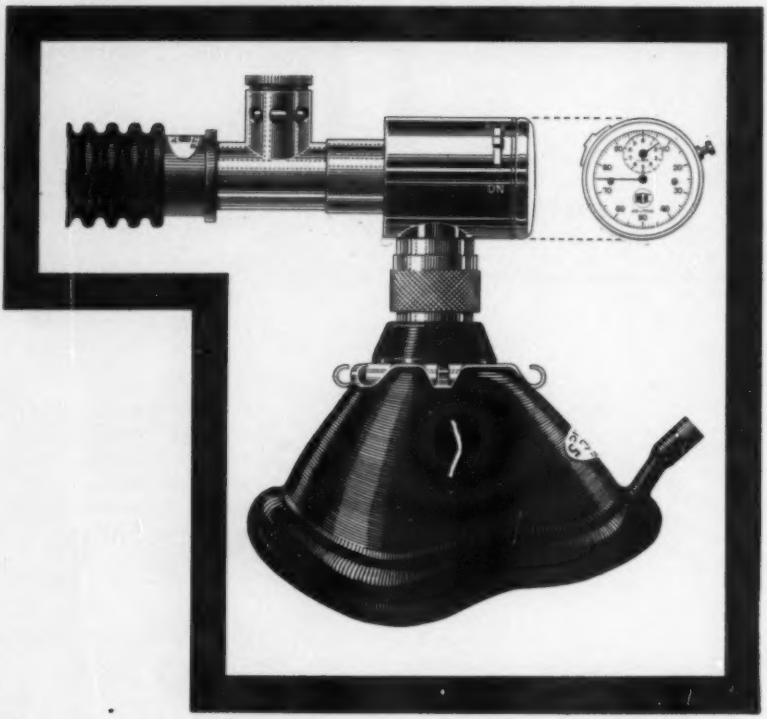
Pressurised with : dichlorotetrafluoroethane and dichlorodifluoromethane.

NET CONTENTS 110 g.

CALMIC

CALMIC LIMITED · CREWE · CHESHIRE
Telephone: Crewe 3251 (7 lines) LONDON: 2 Mansfield Street. Telephone: Langham 8038/9

THE WRIGHT
RESPIROMETER



This is a respiratory measuring instrument for use in anæsthesia, oxygen therapy and for general application in respiratory physiology. It has a watch-type dial which shows at a glance the precise amount of gas that has passed through the instrument. Air resistance and inertia are extremely low, and the instrument can be plugged into any part of an anæsthetic circuit or used with a face mask for respiratory studies. We shall be pleased to send you fully illustrated literature on the Wright respirometer. Just ask us.

BRITISH OXYGEN IN THE SERVICE OF MEDICINE

THE BRITISH OXYGEN COMPANY LIMITED, MEDICAL DEPARTMENT,
SPENCER HOUSE, 27 ST. JAMES'S PLACE, LONDON, S.W.1.

ANNALS
OF THE
ROYAL COLLEGE OF SURGEONS OF ENGLAND

Editor :
SIR CECIL WAKELEY, BT., K.B.E., C.B.,
LL.D., M.Ch., D.Sc., F.R.C.S., F.R.S.E., F.F.R. F.D.S.R.C.S.

Volume 28 JANUARY 1961 No. 1

Published by
THE ROYAL COLLEGE OF SURGEONS OF ENGLAND
LINCOLN'S INN FIELDS LONDON, W.C.2

Annual Subscription - £2 post free
Single copies - - 3s. 6d. post free

ANNALS OF
THE ROYAL COLLEGE OF SURGEONS OF ENGLAND

CONTENTS

JANUARY 1961

		Page
Genetics in Relation to Surgery: A Historical Review	C. E. Dukes	1
Lister Medal		15
Anatomical Museum		15
The Duplicity Theory of Vision	R. A. Weale	16
Faculty of Anaesthetists		35
Bernard Johnson Memorial		35
Natural History of Arteriosclerosis of the Lower Extremities	K. Bloor	36
Admission to the Court of Patrons		52
Appointment of Fellows and Members to Consultant Posts		53
Professor Raffaele Bastianelli		54
The Late Sir Gordon Gordon-Taylor		56
Proceedings of the Council in December		57
Imperial Cancer Research Fund		58
Annual Meeting of Fellows and Members		60
Grant of Fellowship Diplomas		61
Response to the Broadcast by Sir Cecil Wakeley, Bt.		63
Books Added to the Library, July–December 1960		63
Donations		66
Diary for January		68
Diary for February		68

Telegrams : Collsurg, Holb, London
Cables : Collsurg, London Telephone : Holborn 3474

GENETICS IN RELATION TO SURGERY: A HISTORICAL REVIEW

Thomas Vicary Lecture delivered at the Royal College of Surgeons of England

on

27th October 1960

by

Cuthbert E. Dukes, O.B.E., M.D., M.Sc., F.R.C.S.

Consulting Pathologist, St. Mark's Hospital and the Institute of Urology, London

THIS LECTURE COMMEMORATES Thomas Vicary (1495–1561), who led the joint delegation from the ancient Company of Barbers and the Guild of Surgeons when they waited on King Henry VIII, requesting that they might be united into one company. Their request was granted, and in 1540 Thomas Vicary was elected first Master of the new Company of Barber Surgeons.

It is a tradition that this lecture should be on a historical subject of interest to surgeons. Last year the lecture was given by the Master of the Barbers' Company, Mr. Henry Thompson, F.R.C.S., who took as his subject " Sergeant Surgeons to Their Majesties ". This was a historic occasion in more senses than one because it was the first time the Vicary Lecture had been given by the Master during his year of office.

I feel very poorly qualified to follow Mr. Thompson and the long line of his distinguished predecessors, for I am neither a barber nor a surgeon! But for many years I have been privileged to be associated with surgeons in the study of a hereditary disease which is now being treated surgically with increasing success. This work has brought me in touch also with experts in the field of genetics and I have come to realize how much surgeons, pathologists and geneticists may have to learn from each other.

So when I was invited to give this lecture I decided to take as my subject " Genetics in Relation to Surgery " and to draw your attention to some of the surprising ways in which the young science of genetics during its brief history has begun to influence the ancient art of surgery.

Up to the present the most important effect of genetics on surgery has been in relation to the development of blood transfusion, which became a safe procedure as a direct consequence of the discovery of the human blood groups. Looking back now it seems as if by a stroke of good fortune surgery had found a new ally in an unexpected quarter! Somewhat similar in character is the way in which the possibilities and limitations of skin grafting and the transplantation of organs have been clarified by the basic principles of genetics: with hints also of new developments in the future.

So far genetics seems to have had little direct effect on the actual treatment of those hereditary diseases which come within the province of the surgeon, but we must remember that genetics is still a very young science, less than 60 years of age.

From "heredity" to "genetics"

The science of genetics was born in the year 1900. This was the year in which Mendel's work was rediscovered and quoted almost simultaneously in the scientific contributions of three European botanists, one in Holland, another in Germany and a third in Austria (Fig. 1).

Reproduced from "The Life of Mendel" by H. Iltis, by kind permission of George Allen & Unwin Ltd., publishers.

Fig. 1. Gregor Johann Mendel (1822–1884)

The story of this sensational rediscovery has often been told. Mendel's paper had been read to meetings of the Natural History Society of Brünn early in 1865, and was published in the *Transactions* of the Society the following year. The journal describing this work is said to have been widely distributed, and was certainly received in London both by the Royal Society and by the Linnean Society. Yet no attention was paid to it until 34 years after its publication and 16 after Mendel's death.

Mendel's work was commenced in 1857, the year before Darwin and Wallace made their historic communication to the Linnean Society here in London, so Mendel can have known nothing of Darwin's theory of Natural Selection when he first planned his experiments, but by the time he published his work the situation had changed and he recognized that

the study of inheritance had a special importance in relation to evolutionary theory. Later Mendel became a close student of Darwin and in the library of the monastery at Brünn there is a copy of *The Origin of Species* with many notes in Mendel's handwriting.

It seems certain that Darwin never knew of Mendel or his work. No reference to Mendel has been found in Darwin's writing and in the sixth edition of *The Origin of Species*, published in 1872, the following statement was made:

> " The laws governing inheritance are for the most part unknown. No one can say why the same peculiarity in different individuals of the same species or in different species is sometimes inherited and sometimes not so; why the child often reverts in certain characteristics to its grandfather or grandmother or more remote ancestor. . . . " (Colin, 1956.)

Yet these were exactly the questions which Mendel's experiments had answered. Had Darwin come in contact with Mendel's work the history of biology during the last third of the 19th century might have been different.

The prevalent view with regard to heredity throughout this period was that the hereditary qualities of parents blended in their offspring. But if this were so their variety would diminish and ultimately be lost. This presented a difficulty because variation within an animal species was thought to be one of the main causes of evolution. Darwin was aware of this difficulty and suggested that the loss of variation due to blending of characters could be compensated for by the occurrence of spontaneous changes or sports.

Another fresh source of variation might be expected if characteristics acquired by parents could be passed on to their descendants. But evidence of the inheritance of acquired characteristics has always been difficult to obtain.

Meanwhile in the latter half of the 19th century the microscope was coming to play an important part in research into the unsolved problems of heredity. The cell nucleus, its chromosomes and their behaviour in mitosis were accurately described, and before the year 1900 the detailed microscopic study of fertilization and reproduction had provided a mass of information which was soon to serve as a basis for the interpretation of genetics.

I am recalling these facts to explain the enthusiasm with which William Bateson and other biologists were suddenly converted to " Mendelism " when Mendel's work was rediscovered in 1900.

The story of William Bateson's conversion has been graphically described by his wife in her biography of her husband. She says that on 8th May 1900 Bateson was due to give a lecture to the Royal Horticultural

Society on " Problems of Heredity ". The lecture had been prepared, but in the train to London Bateson read Mendel's original paper for the first time. It made such an impression on him that he immediately re-wrote his lecture notes introducing an account of Mendel's work. When writing of this incident more than 20 years later Mrs. Bateson said, " His delight and pleasure on his first introduction to Mendel's work were greater than I can describe: as when with a very long line to hoe, one suddenly finds a great part of it already done by someone else and one is unexpectedly free to get on with other jobs " (Bateson, 1928).

Bateson came to regard Mendelism as a master key in the study of evolution. He later christened the subject " Genetics ", which he defined as " that part of biological science which is concerned with heredity and variation ".

I shall not attempt to trace the development of the science of genetics from its birth to the present stage of its development, a task which would certainly be beyond my capacity, even if I had time. I will only say that in the period immediately following the rediscovery of Mendel's work inherited characteristics were looked upon too much as units and thought to pass from parent to offspring with little regard to the environment. Further investigation soon showed the incompleteness of these ideas, and proved that the gene rather than the characteristic was the unit of heredity.

Genetics and blood transfusion

From this brief review of the origins of genetics let us now pass on to the story of some of its applications in surgery. First in importance is certainly its relation to blood transfusion.

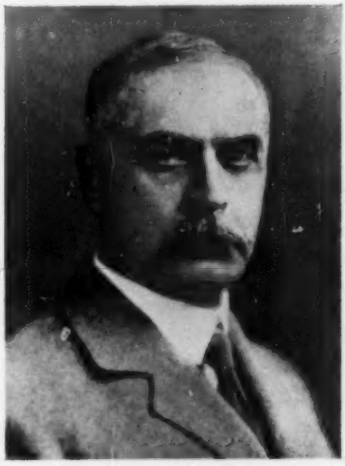
There is no need for me to recall the tragic history of blood transfusion before the application of compatibility tests because this has been so well told by a former member of the Council of this College, Sir Geoffrey Keynes (1949). I will begin with the discovery of human blood groups by Karl Landsteiner (1868–1943) in 1900 (Fig. 2).

It is certainly a strange coincidence that the year 1900 should have witnessed the re-discovery of Mendel's researches on heredity and also the new discovery of the existence of human blood groups, from which both surgery and genetics have profited so much.

The significance of Landsteiner's great discovery was not appreciated at first. He himself may have guessed that the blood antigens might be inherited, but definite evidence of this was not forthcoming until ten years later and another fourteen years were to pass before the genetical analysis of the A.B.O. blood groups was achieved in 1924 by F. Bernstein (Penrose, 1959). This and subsequent genetical researches, particularly in relation to the Rhesus factor, have all contributed to establish blood transfusion as

a safe procedure, far safer than it was when the simple slide compatibility tests made blood transfusions possible in the latter years of the first world war (1914–18).

And of course other branches of medicine have benefited also from research into the genetics of blood groups. I need only remind you of the association of haemolytic disease of the new born with Rhesus incompatibility, a most convincing demonstration of the value of genetics in clinical medicine.



Reproduced from "The Nobel Prize Winners 1901–1937" by MacCallum and Taylor, by kind permission of the Panorama Biographische und Monographische Verslagsgies. m. b. H., Vienna.

Fig. 2. Karl Landsteiner (1868–1943)

Of special surgical interest is the investigation initiated by Professor Ian Aird and his colleagues, which has proved that persons belonging to different blood groups may differ in their susceptibility to certain diseases. Thus people in blood group O have been found to be more liable to suffer from duodenal ulcers than those belonging to other blood groups. On the other hand cancer of the stomach has a higher relative incidence in persons of group A. These are surprising developments, all traceable back to Landsteiner's initial observations on blood agglutination carried out sixty years ago in the Vienna Institute of Pathological Anatomy.

Let us turn back to those days. Landsteiner had recently been appointed as an assistant in the Institute and in his spare time began a research into immunity reactions, a subject in which he was always deeply interested. He took samples of blood from six of his colleagues, separated the serum and prepared saline suspensions of the red cells. He was surprised

to find that these were agglutinated by some sera, but not by others. On the basis of these reactions he distinguished three blood groups, a fourth being discovered two years later by one of his pupils. These are the blood groups now known as A, AB, B and O, though originally designated 1 to 4.

This discovery of the existence of blood groups opened the road to blood transfusion and to Landsteiner alone must be ascribed the credit for this great achievement. None the less on an occasion such as this we should recall that somewhat similar observations on the agglutinating power of blood serum were being carried out at the same time and in this College by the late Dr. S. G. Shattock, F.R.S. (1852–1924).

Shattock made two communications to the Pathological Society of London in 1899, concerning the clumping of a suspension of normal red blood corpuscles by the serum of patients suffering from various diseases. His observations were published next year in the *Journal of Pathology* and in this he illustrated what he had observed when the serum from a patient with pneumonia was mixed with a suspension of normal blood. He considered that this might be similar to the recently discovered Widal reaction, that is to say analogous to the agglutination of bacteria in certain diseases; but if we look at his illustrations in the light of our present knowledge of blood groups it seems possible that the phenomenon he was observing was merely an example of blood incompatibility.

I am not suggesting, of course, that Shattock anticipated or even prepared the way for Landsteiner's great discovery, but only mention it as being of historical interest since Shattock was working in this building at that time.

Karl Landsteiner, universally acknowledged as the discoverer of human blood groups, was born in Vienna in 1868 and died in New York in 1943. In a historical lecture such as this it is appropriate to recall something of the ups and downs of his eventful life.

Landsteiner was thirty years of age when he obtained his first appointment in the Pathology Department of the University of Vienna and remained on the staff for nearly twenty years. Because of personal difficulties he left Vienna in 1919 to take up a post as clinical pathologist to a small Roman Catholic hospital at The Hague, where he worked almost single-handed in a small room with very inadequate equipment, his only assistants being a manservant and a nun. The nun had other duties also in this religious house and forsook the laboratory from time to time to officiate as organist in the hospital chapel.

Yet in spite of all these environmental handicaps, Landsteiner continued his research work throughout his three years in Holland and published twelve papers on immunological problems, five of them in Dutch (Peyton Rous, 1947).

It was only when Landsteiner was invited to the Rockefeller Institute, New York, towards the end of 1921, that for the first time in his life he saw a prospect of being freed from routine duties and of being able to continue his researches in a well equipped laboratory.

When he reached New York in 1923 he was fifty-five years of age, but the foresight and generosity of his American hosts was more than justified because Landsteiner continued his researches with unabated energy and it was in this phase of his life that he began to elucidate the Rhesus factor. Even the award of the Nobel Prize in 1930 did not tempt him to rest on his laurels. He continued at work until 26th June 1943, when he died suddenly from coronary obstruction.

The grafting of skin

I now turn to a field of surgery in the development of which genetics has already begun to stake out a claim, namely the grafting of skin and transplantation of organs. Since this is a historical lecture, I will begin by recalling that in 1597 the famous Italian surgeon Gaspar Tagliacozzi (1546–1599) described a method of repairing defects of the nose which he had carried out with success. The patient's left arm was bound to his head and a flap of skin from over the biceps was attached to the mutilated nose. The poor wretch had to remain in this position for some weeks until the flap of skin, having made its new connections, could be severed from the arm and tucked in round the nostrils.

There was a great need for plastic operations such as this in the 16th century owing to injuries received in duels and street brawls or as punishment for crime: and no doubt many noses were lost also as a result of syphilis.

Tagliacozzi was not the originator of this method of rhinoplasty, but deserves special mention because he recommended that the skin graft should always be taken from the patient and not from another person: in other words that it should be an autograft and not a homograft (Gnudi and Webster, 1950).

Because his successors began to use homografts (skin derived from another person) instead of autografts (skin from the same individual), the operation of rhinoplasty fell into disrepute and Tagliacozzi himself, though he had been honoured and respected by his colleagues during his life, came to be ridiculed, insulted and maligned after his decease. In fact nearly three hundred years were to elapse before his methods were revived by plastic surgeons.

Meanwhile, plastic surgeons learnt from experience that autografts of skin " take " but homografts do not. (There are some important exceptions to this rule, but I will refer to these later.) Plastic surgeons also

learnt that though skin homografts very rarely survive and become permanently incorporated into the body of the recipient, none the less they are often very useful as a temporary expedient. If means could be found to make homografts last longer, or, better still, to take permanently, this would be a very great achievement.

We now know that the explanation why skin autografts " take " and homografts do not is because each individual human being is genetically distinct and unique. Medawar and his co-workers have shown that a graft of skin or any other tissue from another individual, though it may appear to be accepted at first, will eventually provoke an immunological response and be rejected: since it is genetically different it comes to be regarded as of foreign origin (Medawar, 1958). This does not happen with corneal grafts because the cornea is a non-vascular structure and beyond the reach of lymphocytes and other cells responsible for a host reaction to a homograft, but if by accident the cornea should become vascularized the graft will usually fail.

Skin homografts are possible from one uniovular twin to another because such twins are genetically identical: at any rate to the extent of not evoking an immunological response. Also it is possible for skin grafts to be exchanged between some non-identical twins if each twin is a mixture of cells of two genetic origins, the so-called graft-hybrids or chimeras. Another exception is the recently discovered congenital affliction known as agammaglobulinaemia, the victims of which are unable to manufacture the blood protein, gamma globulin, to which most antibodies belong.

These curious exceptions to the general rule are profitable fields for further research because they may perhaps point the way to some device whereby it may be possible to circumvent the biological law which orders the destruction of skin or other living tissue derived from another individual. It is scarcely necessary to add that if the homograft reaction could be overcome or evaded this would result in a great increase in the field of usefulness of skin grafting.

The position with regard to the transplantation of organs is a little different. Here it is important to make a distinction between organs that are expected to continue to live and function in their new home and those which may serve a useful purpose even though they may not remain alive, such as transplants of bone, cartilage or blood vessels.

For transplants in which survival and continued function is essential, such as kidney homo-transplants in man, the situation proves to be even more complex than had been anticipated because W. J. Dempster and his co-workers have shown that such transplanted organs may begin to react against the foreign serum of the host (Dempster, 1953). This threat does not endanger transplants of organs between identical twins, and four years ago Merrill and his associates in Boston reported their first success with a

kidney transplant to a patient with bilateral renal disease who was fortunate enough to have an identical twin willing to donate a kidney (Merrill *et al.*, 1956).

More recently, successful kidney homo-transplantation has been recorded from a non-identical twin, the expected homograft response being averted by pre-operative whole body radiation (Merrill *et al.*, 1960). It has been suggested also that the homograft reaction might be avoided or diminished by the use of cytotoxic drugs, such as chlorambucil. I mention these only as indications of possible ways in which there may be further developments in the future in relation to skin grafting and the transplantation of organs.

I cannot leave the subject of the transplantation of organs without some reference to the great John Hunter, whose name we revere in this College which houses the remains of his world-famous museum. But John Hunter's experiments have been so fully discussed by a former Vicary Lecturer and past President of this College, Sir Cecil Wakeley, Bt., that I need do no more than make a passing reference to his experience in the transplantation of teeth (Wakeley, 1955).

As compared with his contemporaries Hunter seems to have been particularly successful in the transplantation of teeth, largely because when he undertook this operation personally or superintended it in others he chose the donor with special care and was exceptionally skilful in his technique. He believed that transplantation of teeth from donor to recipient was possible because of " a disposition in all living substances to unite when brought into contact with one another ". I will only add that Hunter would have been quick to grasp the new conception of our physical constitutions which the study of genetics has revealed and would have been filled with wonder at the contribution of this new science to surgery.

If fifty years hence some future Vicary Lecturer should again take as his subject " Genetics in Relation to Surgery ", I hope he will not fail to mention an incident of our own times when skin grafting was undertaken by the late Sir Archibald McIndoe to settle a problem of disputed paternity (McIndoe and Franceschetti, 1950). This literally " made " history. Here is the story:

Three children Pierre, Victor and Eric were born in the same clinic on the same night. Pierre and Victor were regarded as twins and handed over to one set of parents and Eric to another. Six years later the supposed father of both Pierre and Victor met Eric, who seemed to him to be the very image of his boy Victor. It seemed possible then that Victor and Eric were the twins and that the children had somehow been muddled up. Character comparisons and blood tests did not settle the uncertainty, and in this predicament Sir Archibald McIndoe was asked to perform a test

by which the case would be agreed to stand or fall. Small skin grafts were exchanged, on the one hand between Pierre and Victor, and on the other hand between Victor and Eric. The skin grafts exchanged between Pierre and Victor were destroyed and sloughed away in a matter of weeks: those exchanged between Victor and Eric survived.

On the basis of these tests Pierre's mother was willing to accept Pierre as her son and Eric, the misplaced twin, moved to his new home to be united with his twin brother Victor.

This application of skin grafting as a test of disputed paternity proves not only that surgery has gained from genetics but also that it has something to give in return.

Genetically determined surgical diseases

I now come to the influence of genetics in the treatment of surgical diseases known to be genetically determined.

It must be admitted at once that so far this young science during the first sixty years of its existence has had little direct effect on the treatment of hereditary diseases by the methods of surgery. But genetics soon began to influence the development of pathology, especially in biochemistry and cytology, and research work in these fields has sometimes indirectly been of interest to surgeons. Also the clinical experience of surgeons has sometimes led to new discoveries in genetics. For instance the observation that certain individuals were unduly sensitive to suxamethonium led to the recognition of inherited differences in the formation of the enzyme pseudocholinesterase (Harris, 1959).

The recent discovery that some of the so-called " inter-sex " anomalies may be directly related to abnormalities in chromosomes is certainly of surgical interest because it is generally agreed that " inter-sex " patients, whether true hermaphrodites or pseudo-hermaphrodites, should be brought up in the sex to which they have been accustomed and to which superficially they appear to belong. In order to achieve this it may be necessary for the surgeon to remove evidence of the other sex. In the future, therefore, the geneticist may be called in to " give evidence " when surgical treatment is to be considered (Gordon *et al.*, 1960).

You would not expect me to go further into these recent exciting developments. This is a historical survey and need not include " stop-press " news!

Returning once more to the past, let me remind you that in the pre-genetical era the presence or absence of a hereditary factor in disease was based exclusively on the family history, and by this means alone a great deal about the inheritance of disease was learnt during the 18th and 19th centuries. For instance, most of the essential facts about the inheritance

of haemophilia were recorded by Dr. John C. Otto of Philadelphia in 1803. He gave the first accurate account of what he described as " a haemorrhagic disposition existing in certain families ". He noticed that it occurred only in males but that not all were affected. He drew attention also to the remarkable fact that females did not suffer from the disease, but that they might transmit it to their children (Bullock and Fildes, 1911).

This kind of careful recording of what could be observed to pass from one generation to the next remained the only way of observing the hereditary factor in disease until the rediscovery of Mendel's work led to the possibility of distinguishing both " dominant " and " recessive " types of

Reproduced by kind permission of the Royal Society.

Fig. 3. Sir Archibald E. Garrod (1857–1936)

inheritance. Many instances of what we now call dominant inheritance were previously known but the first example of recessive inheritance was recorded by A. E. Garrod in 1902 (Fig. 3).

Garrod (later Sir Archibald Garrod) (1857–1936) in his investigation into alkaptonuria and other inborn errors of metabolism had noticed that these might affect two or more of a group of brothers and sisters though the parents themselves were normal. He noticed also that the parents of alkaptonurics were often blood relations, usually first or second cousins. At this point he consulted with William Bateson, who pointed out that the situation might be readily explained by the recently discovered laws of Mendel, because what Garrod had observed was precisely the type of familial distribution to be expected if alkaptonuria was inherited as a Mendelian recessive character.

Thus Garrod's epoch-making study of inborn errors of metabolism not only stimulated much further research in biochemistry but also had the effect of increasing the importance of a patient's family history and indeed increased its scope so as to include information concerning consanguinity of parents and the possible occurence in the patient or relatives of other abnormalities. Subsequent discoveries in genetics have indicated that the family history may need to be further supplemented in some cases by biochemical and haematological investigations and by tissue culture and chromosome analysis.

To illustrate the value of co-operation between surgeons, pathologists and geneticists I must ask to be allowed to refer to a hereditary disease of interest to surgeons in which I have had an opportunity of gaining special experience, namely familial polyposis of the colon. This came to be recognized as a distinct entity towards the end of the 19th century. At this time its liability to be associated with intestinal cancer was not generally known. The all-important fact that the polyposis *preceded* the cancer was not definitely established until the sigmoidoscope came into use early in this century.

My experience of this disease dates from the year 1925, when the late Mr. J. P. Lockhart-Mummery wrote an article for the *Lancet* entitled " Cancer and Heredity " in which he described three polyposis families in each of which there was a high incidence of intestinal cancer. I have kept in touch with these families since then, and with many others, and now have records of more than eighty families in which this disease has appeared.

For polyposis patients who require major surgery there are at present only two forms of treatment: either excision of the whole large bowel and performance of permanent ileostomy or preservation of part of the rectum and anastomosis of the ileum to it. Experience has shown that this second form of treatment, ileo-rectal anastomosis, gives excellent results, but it cannot of course be applied in those patients who already have a carcinoma of the rectum, nor in those in whom the polyps are so numerous in the rectum that their destruction by diathermy would be likely to result in much scarring and subsequent stenosis.

The great advantage of ileo-rectal anastomosis is of course that it avoids a permanent colostomy, and to illustrate the value of this operation I will show you the pedigree of a polyposis family in which all the six affected children in the fourth generation have been treated successfully in this way as soon as the severity of the symptoms made major surgical treatment necessary (Fig. 4).

Five of these (IV. 2, 3, 5, 6, 7) were operated on by Mr. C. Naunton Morgan and one (IV. 1) by Mr. H. E. Lockhart-Mummery.

I have known this family for many years and stood in the background, so to speak, doing little more than record the tragic story as it has slowly unrolled itself from year to year. But, standing there in the background, I have also witnessed the unsparing devotion and unfailing skill of their surgeon, Mr. C. Naunton Morgan, and what finer tribute could there be to this than the simple fact that, after all these many years, the widowed mother and all her children still regard Mr. Naunton Morgan as their best friend in the world!

The eighty families which are being followed up in the research department at St. Mark's Hospital now include more than a hundred children of affected members and an attempt is being made to keep in touch with all of these so that those who are destined to develop the disease may receive surgical treatment as early as necessary (Dukes, 1958).

All the surgeons on the staff of the hospital have cooperated in this research, especially, during recent years, Mr. Henry Thompson and Mr.

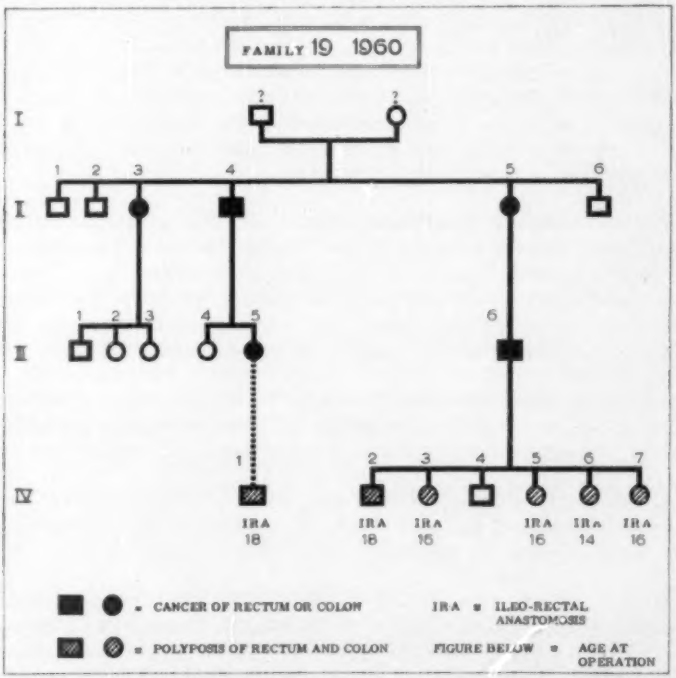


Fig. 4. Familial Intestinal Polyposis. Direct descendants of affected members only recorded. All the six affected children in the fourth generation have been treated by colectomy and ileo-rectal anastomosis.

H. E. Lockhart-Mummery, both of whom have been tireless in their efforts to trace relatives of polyposis patients and to examine them, either by visiting their homes or by special appointments at hospital.

This work provides a good example of cancer control—in fact it would be difficult to find a more promising field for the exercise of cancer control than a polyposis family, because both diagnosis and treatment are possible in the precancerous stage of the disease and because the results of surgical treatment are so good. Of course there are still many unsolved problems in the genetics of polyposis and in the study of these we now have the advantage of help from Dr. Arthur Veale from New Zealand, who is working also under the supervision of Professor Penrose at University College.

Here, then, is an example of a disease in the study of which surgeons, pathologists and geneticists are advancing together. As proof of the progress already made let me recall that thirty years ago patients who had inherited polyposis seldom came to hospital for treatment until cancer had supervened. They were relatively young victims, 20 or 30 years younger than other patients with colon or rectal cancer. After surgical treatment, even at its best, the prognosis for them was poor. In those days much less was known of the mode of transmission of the disease, and in families with many affected members the threat of cancer seemed to overshadow each one like an inescapable doom.

Contrast this with the situation today. First, in most cases diagnosis by means of the sigmoidoscope and X-rays is possible even before the onset of symptoms. Secondly, if surgical treatment becomes necessary and is carried out at an early stage, it will often be possible to undertake this without resulting in a permanent colostomy or ileostomy. Third, and most important of all, the risk of cancer is greatly decreased.

I have often reflected that the pious Mendel would have been much surprised to learn that knowledge gained from his experiments with the garden pea would, a century later, prove useful in the study of polyposis.

It was of course because I have seen the value of cooperation between surgeons, geneticists and pathologists that I chose as my subject today a historical review of the relationship of surgery and genetics.

It only remains now for me to say to you, Mr. President, and the Council of the Royal College of Surgeons and the ancient Company of Barbers that I am indeed most appreciative of the great honour you have conferred on me in appointing me to give the Thomas Vicary Lecture this year, and to express the hope that you may consider it worthy of the occasion.

REFERENCES

BATESON, B. (1928) *William Bateson, F.R.S.* Cambridge.
BULLOCK, W., and FILDES, P. (1911) *Treasury of Human Inheritance*, Parts 5 and 6, Sect. XIV, p. 169.
COLIN, E. C. (1956) *Elements of Genetics: Mendel's Laws of Heredity with special application to Man*, p. 5. New York, Toronto, London.
DEMPSTER, W. J. (1953) *Brit. J. Surg.* **40**, 447.
DUKES, C. E. (1958a) *J. Roy. Coll. Surg. Edin.* **3**, 182.
————(1958b) *Dis. Colon Rectum*, **1**, 413.
GNUDI, M. T., and WEBSTER, J. P. (1950) *The Life and Times of Gaspare Tagliacozzi.* New York.
GORDON, R. R., O'GORMAN, F. J. P., DEWHURST, C. J., and BLANK, C. E. (1960), *Lancet*, **2**, 736.
HARRIS, H. (1959) *Human Biochemical Genetics.* Cambridge Univ. Press.
KEYNES, G. (1949) *Blood Transfusion.* Bristol.
LOCKHART-MUMMERY, J. P. (1925) *Lancet*, **1**, 427.
MCINDOE, A., and FRANCESCHETTI, A. (1950) *Brit. J. plastic Surg.* **2**, 283.
MEDAWAR, P. B. (1958) *Proc. Roy. Soc.* **149**, 145.
MERRILL, J. P., *et. al* (1956) *J. Amer. med. Ass.* **160**, 277.
———— (1960) *New Engl. J. Med.* **262**, 1251.
PENROSE, L. (1959) *Outline of Human Genetics.* London.
ROBERTS, J. A. F. (1959) *An Introduction to Medical Genetics.* 2nd edition. London.
ROUS, P. (1947) Karl Landsteiner. *Obituary Notices of Fellows of Royal Society*, **5**, 295.
SHATTOCK, S. G. (1900) Chromocyte Clumping in Acute Pneumonia. *J. Path. Bact.* **6**, 303.
WAKELEY, Sir Cecil (1955) *Ann. Roy. Coll. Surg. Engl.* **16**, 69.

LISTER MEDAL

THE LISTER MEDAL for 1960, which is awarded in recognition of distinguished contributions to surgical science, has been granted to Professor Wilder Graves Penfield, O.M., C.M.G., M.D., Hon. F.R.C.S. England and Edinburgh, Director of the Montreal Neurological Institute, in recognition of his service to the advancement of medical and biological knowledge, especially in the fields of neurology, surgical neurology, and psychology; and equally as humane surgical healer and as scientific researcher. Professor Wilder Graves Penfield will deliver the Lister Memorial Lecture in London on 5th April or date to be arranged in 1961, at the College.

This is the 13th occasion of the award, which is made by a Committee representative of the Royal Society, the Royal College of Surgeons of England, the Royal College of Surgeons in Ireland, the University of Edinburgh and the University of Glasgow.

ANATOMICAL MUSEUM

THE SPECIAL DISPLAY for the month of January consists of photographs of instruments and other personal relics of John Hunter, most of which were lost during the bombing of the College.

THE DUPLICITY THEORY OF VISION

Edridge-Green Lecture delivered at the Royal College of Surgeons of England

on

28th March 1959

by

R. A. Weale, Ph.D., D.Sc.

Institute of Ophthalmology, London

BY ASKING ME to pay tribute to the memory of the late Frederick William Edridge-Green, this College has greatly honoured the Visual Research Division of the M.R.C.'s Ophthalmological Research Unit and I return you many thanks for this distinciton.

The delivery of a Memorial Lecture is an experience of a lifetime. Nor is it an everyday matter to address an expert audience in this great hall where august names surge on and up all along the walls. If, then, I fail in my expression of thanks for the honour you have done us, it is because, though not a girl, like Portia, I am unlessoned, unschooled, unpractised.

I say: unlessoned, unschooled, unpractised; but not unread. I can, therefore, try to examine the relation of Edridge-Green's views to theories of vision as a whole. For we have to remember that Edridge-Green's claim to recognition—had he made one—would have been based less on the invention of his lantern or on his ideas regarding colour vision than on his contribution to the general physiology of vision. This, of course, is the excuse for the title of my talk to you to-day.

The first explicit statement of the duplex nature of our vision appears to occur in Goethe's Theory of Colour. But it is hinted at even by Francis Bacon, who writes in his essay on the Unity in Religion: " all colours will agree in the dark ". We infer that colours do not agree in the light. And Goethe writes: " Accordingly as it is affected by light or dark, the retina exists in two totally different states, wholly opposed to each other. They are characterized by extreme insensitivity and sensitivity respectively." Etc., etc.

It is noteworthy that Goethe and Purkinje were in close correspondence. Goethe accused Purkinje of not according him priority regarding the discovery of some important visual effects, a complaint which has become standard practice in visual physiology. Later Purkinje apologized and explained his position to Goethe, the two becoming firm friends, a circumstance which has not, however, become a precedent in this subject.

You remember that Goethe represents colours as steps between darkness and light, a concept which stamps that poet with the mark of Neo-platonism just as surely as that school can claim Dante because of the stratification he imposed on the road from Hell to Heaven. It is, I feel, an interesting speculation that we may owe one of the most catholic generalizations in the study of the eye to a school of philosophy in the first place, and only secondarily to direct observation.

The present-day statement of the duplicity theory, however, is rooted in the work of Schultze, Parinaud and von Kries. Schultze, one of the earliest and greatest retinal anatomists, related retinal structure to animal behaviour, and concluded that rods are associated with nocturnal but cones with diurnal habits. Parinaud and von Kries supported this view with evidence based on human sensory physiology and pathology, and came to summarize the theory as follows:

Daylight or photopic vision is mediated by the cones, night or scotopic vision by the rods (Fig. 1).

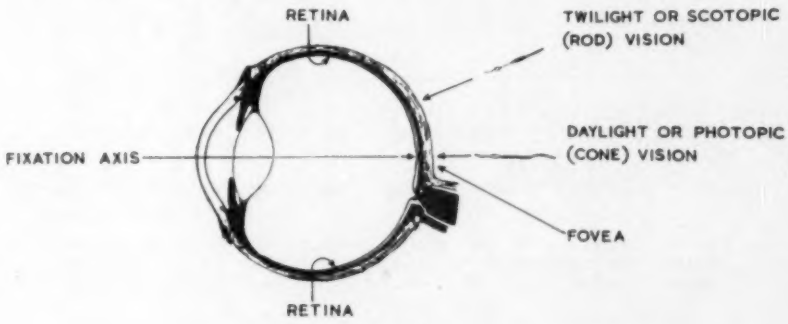
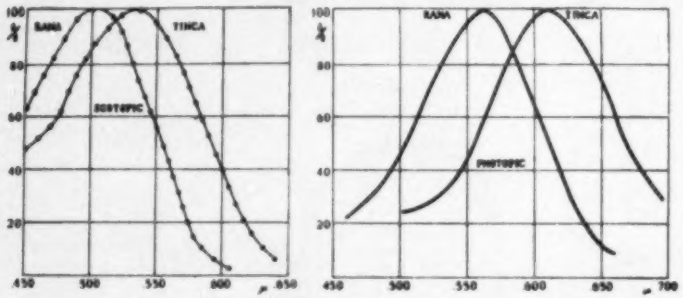


Fig. 1.

You can divide writers dealing with this theme into stalwarts and diplomats. The stalwarts believe that duplicity is rigorous. They hold that as light increases in intensity rod activity fades out and cone activity fades in if the eye is blessed with both. Rod activity, characterized photochemically by visual purple, may be inhibited by cone activity; but in the absence of cones the animal becomes photophobic and/or goes to sleep during the day. Again, because our own cones mediate colour vision, the stalwarts *a priori* incline to the view that the mere existence of cones implies that their owners differentiate colours. The idea that vision is dichotomous finds support in the electro-physiological researches of Granit (1947). You may remember that he illuminated various animal retinae with lights of different colours. He found that the threshold intensities required to produce an electric discharge in the

ganglion cells which he tapped follow two characteristic spectrum curves accordingly as the eye is light or dark adapted (Fig. 2). Because the mechanisms cover a large part of the spectrum, Granit called them dominators. He distinguished them from the narrower modulators (Fig. 3), believed to underlie potential mechanisms for colour vision. Granit's researches reveal a dichotomy as regards general brightness responses, governed by scotopic or photopic dominators, without prejudice to the question whether finer analysers, such as modulators, are present or not.

Wald, of Harvard, is another stalwart. Brought up by the puritanical Selig Hecht, he failed to produce the sort of reaction which—in happier days—would have been called the period of restoration. On the contrary, he has apparently demonstrated the universality of duplicity in the chemical substrate of vision. The idea underlying his scheme is impressive in its



Reproduced by permission.

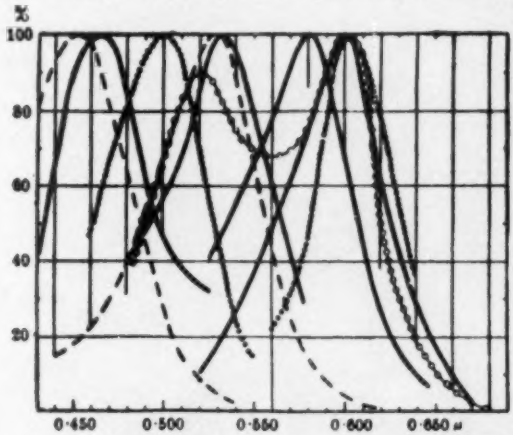
Fig. 2. Comparison of the spectral sensitivity curves, recorded electrophysiologically from frog and tench retinae. Left: dark-adapted, right: light-adapted. Abscissa: wavelength (violet on left, red on right); ordinate: relative sensitivity. (Granit, 1947.)

simplicity. The visual pigments contained in the rod and cone outer limbs are made up of complicated molecules which consist essentially of two parts, namely the chromophore and the carrier. Known chromophores are derivatives of vitamin A_1 or A_2 but this is not where the duplicity lies. The dichotomy consists in the carriers or opsins. Wald (1958) thinks that they are of two sorts only, namely photopsins and scotopsins. By isolating chromophores and opsins and suitably incubating one with the other, Wald has succeeded in synthesizing light-sensitive compounds similar to those directly extractable from animal retinae. He even synthesized a pigment which has not so far been extracted from any retina and which he calls cyanopsin. This latter—a mere test-tube baby—he believes to be the cone pigment responsible for red colour responses much as he believes iodopsin (obtainable from chicken retinae) to underlie green colour responses. I think the fact that more recent work done by Dartnall (1960), by Rushton (1958), and by myself (1955, 1959) fails to

lend credence to some of the conclusions which Wald draws from his experiments, detracts little from the excellence of his studies although it challenges the design which underlies them.

I should now like to mention some members of the diplomatic corps, namely those who believe that the indubitable morphological dichotomy of the human retina is blurred physiologically either by mixed or shared sensory activity or—as in the case of Edridge-Green—by a fundamental difference in function.

Edridge-Green (1920) maintained that a rod acts as a gland which secretes visual purple upon illumination. The visual purple, which he emphasized he had seen suffusing the foveal cones, stimulated them and



Reproduced by permission.

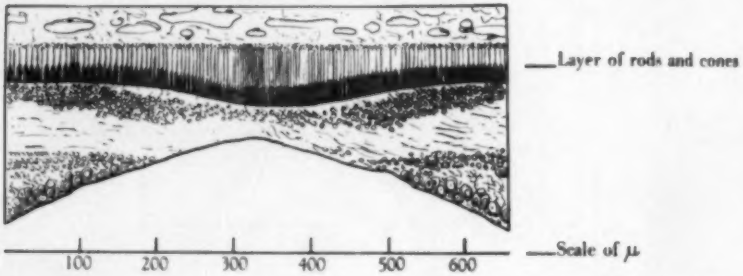
Fig. 3. Modulator curve obtained from a variety of animals. Abscissa and ordinate as in Fig. 2 (Granit, 1947).

thus initiated the visual impulse. This interesting hypothesis cuts right across the orthodox duplicity theory—which, as we recall, ascribes visual functions to both rods and cones—and has been rigorously examined by Sir John Parsons (1924). While perhaps a little problematical in its finer detail, Edridge-Green's hypothesis is of general interest because it attempts to explain the whole of vision in terms of only one visual pigment. He believes that visual purple, modified by its products of bleaching when the eye is subjected to daylight illumination, may well be capable of mediating daylight or photopic vision. This idea of sensory duplicity through pigmentary unity was examined more recently with exquisite mathematical finesse by Dartnall (1948) and qualitatively by Berger and Segal (1950). Dartnall argued that at photopic, i.e. daylight, levels of illumination a fair amount of visual purple could be changed into a secondary product. The combined absorption properties of the parent and daughter substances

reasonably approximate to the spectral characteristics of daylight vision. I seem to be the only person in the world to believe that Dartnall's theory is fundamentally inescapably correct although we now know that its potential applicability is more limited than might have appeared to be the case some time ago.

Willmer is another diplomat. He believes that, while the rods fulfil their orthodox function at low levels of illumination, they mediate our sensation of blue when the level of illumination is raised. They may do so: but I think there are weighty histological objections to the view that they must do so in the fovea. However, I should not be at all surprised if rods contributed to a blue response syste. 1 in the periphery.

At this juncture it may be interesting to examine more closely in what respects rods and cones are believed to differ, especially in regard to their function as scotopic and photopic elements respectively. Let me show



Reproduced by kind permission.

Fig. 4. Section through the central fovea of man (after Thomson and Wright (1947) *J. Physiol.* **105**, 316).

you, in the first place, a table which will, I hope, illustrate my point. I would remind you only that the normal region of visual fixation, the central fovea (Fig. 4), is entirely free of rods, and contains only cones. The perifoveal regions, while not free from cones, are certainly dominated by rods. Hence we can, under carefully selected conditions, stimulate either only cones, or very nearly only rods.

Some of the properties of the receptors can, of course, be studied by probing animal retinae. We owe to them much of our knowledge, but also quite a few mistakes. However, the duplicity theory is not believed to apply to man alone so that in a survey of its fundamentals we can step over species differences provided we do not stumble over them.

This is a curtailed representation of the orthodox story. It is, of course, impossible fully to examine it, but a few of these points are illustrated in some animated slides for which I am indebted to Dr. Peter Hansell and

TABLE

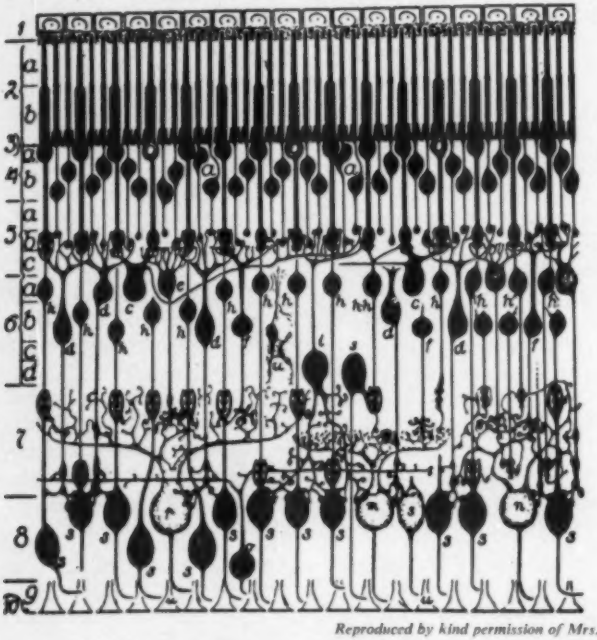
	Rods	*Cones*
Number	120,000,000	6,000,000
Activity	dim light	daylight
Visual acuity	poor	good
Sensitivity	high	low
Rate of dark adaptation	slow	rapid
Maximal sensitivity	blue-green	yellow-green
Colour vision	absent	present
Stiles-Crawford effect	absent	present
Specific destruction by iodo-acetate	yes	no
Amount of visual pigment ..	high	low
Miosis	poor	ready

Mr. T. Tarrant. They depict the duplex nature of our retinae from the functional, photochemical and sensory point of view. E.g. we shall recall that the pupil constricts practically only when light strikes the fovea. We shall see that the light-absorbing properties of the fovea differ from those of the periphery. This leads to the colour shift, named after the celebrated Purkinje, who observed that, in the dark, our eyes are sensitive mainly to blue and green, but in full light to yellow and orange. We shall see further how the fovea needs a lot of light to stimulate the optic nerve because it badly adds up illumination of individual cones. But the periphery needs less light because many rods can add stimuli and reach one and the same optic nerve fibre (Fig. 5). Then, to face the problem of dark-adaptation, we shall remind ourselves that when the eye has been exposed to a bright flash, foveal or central vision is the first to recover from the effects of glare. And we shall ask ourselves which of several possibilities can help our understanding of what happens when the eyes recuperate in the dark. Is it that the connections between the neural elements alter in some way or is it that bleached substances in the rods and cones are re-formed in darkness?

And now I should like to single out two points with which I have concerned myself during the last eight or nine years. They deal with the sensitivity of the human eye and the concentration of visual pigments both in animal and human retinae.

Until a few years ago the situation regarding the relation between sensitivity and visual pigment concentration was simple. Extracts made of the light-sensitive contents of mixed mammalian retinae—i.e. such as contain both rods and cones—failed to reveal the presence of anything but rod visual purple. Granted that Dartnall's brilliant analytical techniques had broken through the one-pigment barrier in fish and amphibia with well-defined and reproducible results, in mammals, retinae histologically heterogeneous were always homogeneous when examined for their photochemical contents: only rod pigments admitted being present. But in order to see we must absorb light. We can see with cones—and how!—therefore they must be able to convert light energy into a neural

message. But where is the absorber? Visual physiology here came to the rescue of visual photochemistry : is not the rod threshold a thousand times lower than the cone threshold? And if that is so, would one not expect cone pigments to be present in the eye in such minute quantities as to escape detection even with present-day techniques? Wald described the situation with epigrammatic elegance only in 1954: " Large as rhodopsin looms in the microstructure of a rod, it occupies a correspondingly small position in the microstructure of a cone . . . A cone, which



Reproduced by kind permission of Mrs. Polyak.

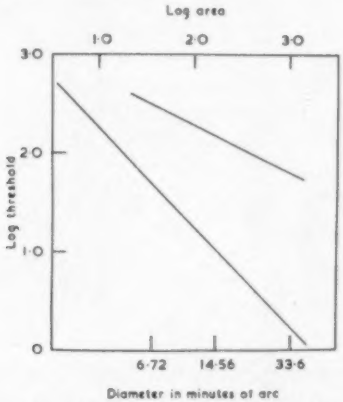
Fig. 5. Schematic section illustrating nervous interaction in the retina (Polyak, *The Retina*, Univ. of Chicago Pr. 1941).

possesses very little visual pigment and hence a small probability of absorbing light, has a correspondingly high threshold."

Such considerations, however, by-passed facts which were beginning to get well established. The first of these—and one often ignored at this very hour—is the futility of discussing visual sensitivity if we disregard the extent of the stimulated area. Obviously, it is not the area which matters so much as the total number of illuminated receptors. In other words, we have to consider whether summation of impulses occurs. A photographic film can perform temporal summation, i.e. if we expose it with a

given intensity for a given time we can, within limits, double the intensity and halve the time, and still get the same result. But the film will fail with spatial summation: we do not get the same result with a given light intensity spread over a thousand grains in the emulsion as when we double the intensity and use five hundred grains. The retina, however, can do both: within limits it will integrate impulses both spatially and temporally.

But there are marked differences as between foveal and non-foveal regions. Leaving temporal summation aside, the fovea is much worse at summating than is the periphery. This is illustrated by Baumgardt's data (1949a), which suggest that extra-foveal areas pool the stimulation of the retinal receptors and conduct them to a final common path much more readily than does the fovea (Fig. 6).



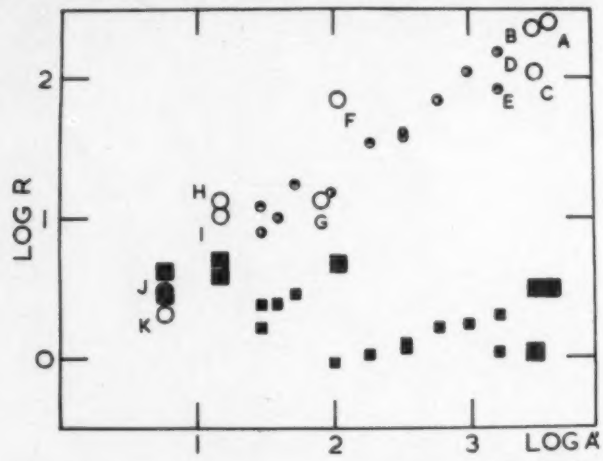
Reproduced from "Comptes Rendus des Seances de la Societe de Biologie" (1949) **143**, *786, by permission of the author, E. Baumgardt.*

Fig. 6. Relation between absolute threshold and size of test-field. Upper zone: fovea. Lower zone: periphery.

I think Stiles and Crawford (1937) were the first to indicate the importance of the size of retinal area. Hecht and others who used a 2° field, i.e. one subtended by a penny at a distance of one yard, found that rods were a few hundred times more sensitive than cones. In Stiles and Crawford's work this factor was reduced to barely ten. The reason for this apparent discrepancy lies in their use of a minute test-field only 4′ of arc in diameter, i.e. one corresponding to a penny at a distance of 30 yards. We see, therefore, that the factor by which rod sensitivity exceeds cone sensitivity depends on the size of the test-field which we employ. This is confirmed by another experiment of Baumgardt's (1949b) in which he measured the ratio of the two thresholds for various sizes of test areas. The rods are found to be 80 times as sensitive as the cones when the area subtends at the

pupil an angle of 45′ of arc, but only eight times as sensitive at the smallest areas. The results obtained by other authors fit into this scheme very well indeed (Fig. 7).

It follows from these data that when small retinal areas are illuminated it does not matter very much whether they are dominated by rods or by cones: the two thresholds are of the same order. We see, therefore, that we are not entirely justified in quoting differences between rod and cone thresholds in support of the Duplicity Theory. You may well say that this variation with area is only one concept and a phoney one at that. Why do the large areas support the orthodox view of the Duplicity Theory?



Reproduced by permission

Fig. 7. Ratio of foveal to peripheral threshold (ordinate) as a function of size of test-field (abscissa). White symbols: experimental data. Black symbols: ditto when receptor density and neural summation are allowed for. Small symbols (D and E) due to Baumgardt (1949b). (Weale, 1958.)

The answer is, I think, as follows: The foveal cones, capable of little summation, converge onto a given optic nerve fibre in much smaller numbers than do peripheral receptors. Now the object of light absorption is not so much the setting up of a discharge in the receptor layer as in the optic nerve fibres themselves. It may happen, therefore, that the light absorbed in any one of a fair number of foveal cones is too weak to produce a discharge which the cortex will consider as worthy of the concept of light. However, the same amount of light absorbed in an equal number of rods will give rise to a sensation of light. The reason is that the rods have been able to pool the discharges set up in them for they converge onto a smaller number of optic nerve fibres than do an equal number of foveal cones. A military comparison will help to illustrate this. If, on a wide

front, tank faces gun, and gun tank, there will take place a to-ing and fro-ing without a real response. But let one side converge its power onto a small part of the enemy front and a break-through will ensue. This ability to converge receptor discharges onto the final common path can be expressed quantitatively (Fig. 6). It is found, for example, that doubling a peripheral area enables us to reduce by 50 per cent. the intensity required to reach threshold stimulation. But in the fovea the advantage amounts to only 15 per cent. The gain obtained can be measured and expressed by the so-called index of summation. I do not wish to bandy about algebraic symbols, but should like to show you merely the result of a few calculations. Thresholds obtained with larger areas both in the fovea and in the periphery can be analysed with the index of summation. Moreover, the number of receptors involved in the measurements can be estimated. When this is done on the data I showed you previously all the results are found to fall approximately into line (Fig. 7). We can, therefore, advance tentatively three important conclusions:

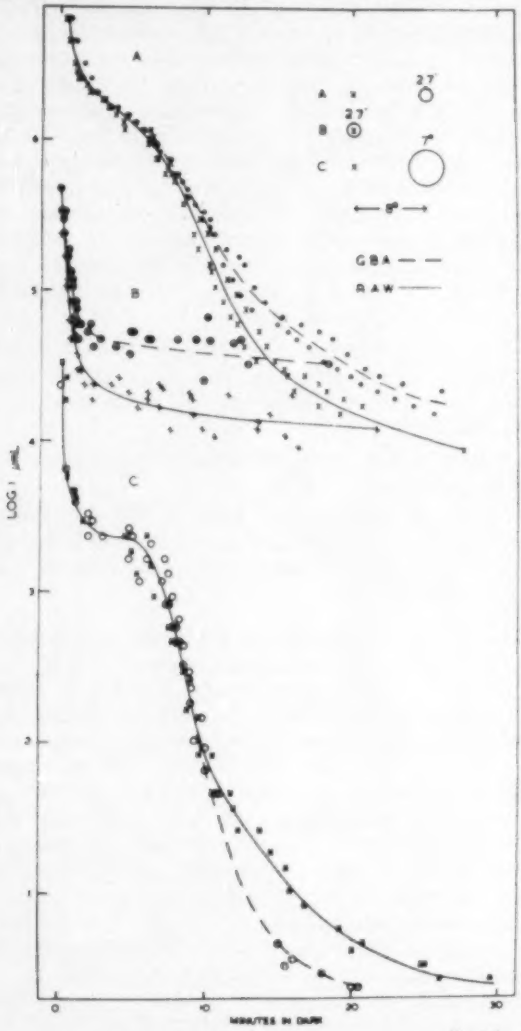
(1) When retinal summation is allowed for either experimentally by using small fields or by calculating the index of integration, the ratio of rod threshold to cone threshold is approximately independent of the number of receptors stimulated.

(2) Under these conditions, single rods and single cones are approximately equally sensitive.

(3) The advantage which large numbers of rods gain in the course of dark-adaptation over equal numbers of foveal cones is due primarily to changes in the interaction of neurones associated with the rods, and not to the rods themselves.

This is illustrated in a study of dark-adaptation, which I carried out with Dr. G. B. Arden. We used a very large and a very small test-field respectively (Fig. 8). The curve C is perfectly orthodox. The early part is due to the increase in sensitivity of the peripheral cone mechanism, and the later to the increase in sensitivity of the rod mechanism. The same is true of curve A. There are, however, two important differences between these two sets of data. In the first place, the difference between the cone and rod final thresholds is much larger in the large field data than in those for the small field. This illustrates the fact that rods can join sub-threshold responses more readily even than peripheral cones; one might say, I hope with impunity, that large numbers of cones suffer from more inhibitions than do rods. The second point I should like to emphasize is that in curve C the final rod threshold is approached much more rapidly than it is in curve A. There is no reasonable photo-chemical process which can be made wholly to account for either of these observations. We are forced to return to the thesis which I put to you, namely that the post-receptoral structures change their ability to pool the rod responses. As the rods become steeped in darkness their inhibitions

disappear. Why this should take place at the speed here indicated is still unknown: a vague but plausible explanation is that an inhibitor substance is gradually dispersed when the eyes are left to themselves. It has been



Reproduced by permission.

Fig. 8. Dark-adaptation curves measured for two observers with large and small test-fields, in the fovea and periphery (Arden and Weale, 1954).

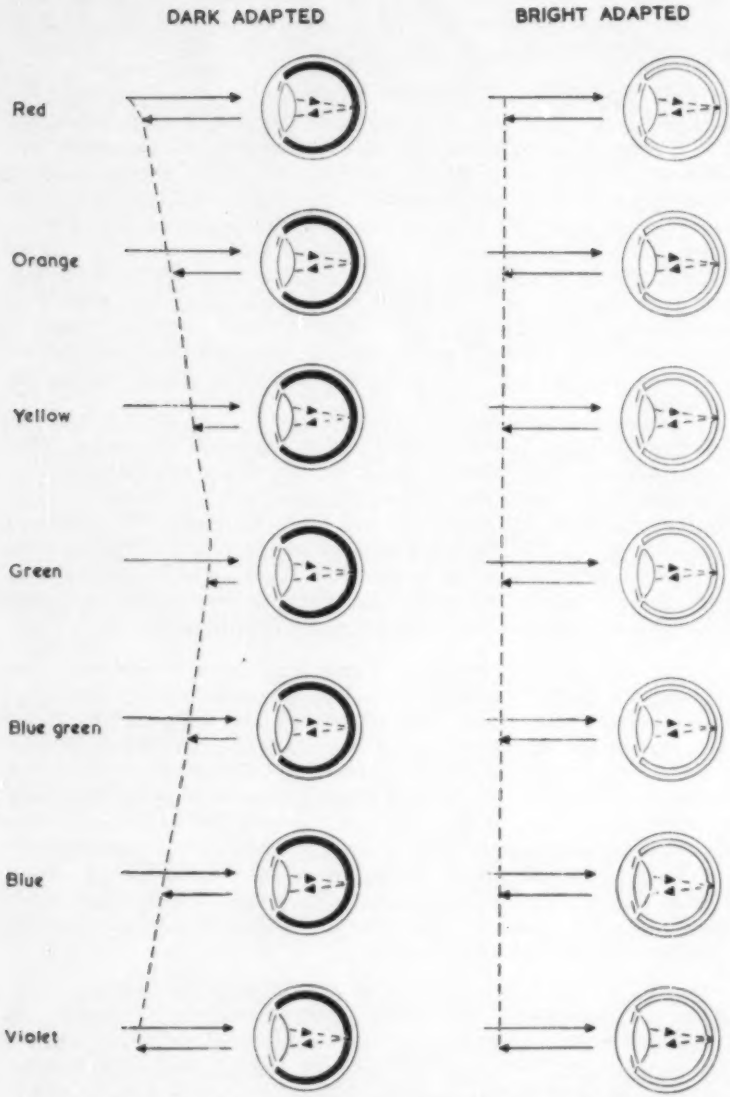
argued that the speed of this increase in pooling—or of the increase in the receptive retinal field—is very slow: I think it is a beautiful example of homeostasis and shall comment on it later on.

These data show something else. Like Professor Wald (1958), you may have noticed that the small-field results (curve A) belie my claim that rods and cones are approximately equally sensitive when summation is allowed for. Is it not a fact that the cone threshold is about a hundred times higher than the rod threshold? Let me remind you that in the retinal region concerned there are about ten rods to every cone. On this count alone, therefore, the rod advantage of 100 reduces to 10. If we allow furthermore that there is probably only one type of rod in the human periphery, but there are three cone types only one of which is stimulated, the advantage is reduced by another factor of three. You see, therefore, that so far from demolishing the case for the approximate equality of single absolute rod and cone thresholds, these data actually support it.

As a comment on these views I would say that they do not shake the Duplicity Theory. But they introduce a change of emphasis. They suggest that, in thinking about the Theory, we should use the terms rods and cones merely as abbreviations: for what appears to matter is the mechanisms which are associated with these receptors. The rods and cones are the aerials: the work is done by the power supply and the amplifiers behind them. Just as crystal sets went out of fashion a year or two ago, so I feel that we should start ascribing less importance to rods and cones and consider rather the associated systems as a whole.

However, I have not finished. I mentioned how—within the framework of the Duplicity Theory—visual physiologists came to the rescue of the photochemists; how when the latter found rod pigments but no cone pigment the physiologists saved them by pointing out that cones have a much higher threshold than do rods. But I hope to have shown that this difference in threshold is illusory, and due to retinal organization, not to receptor structure. If correct, this would mean that we face a dilemma for Wald's concord between the relative concentrations of photopigments on the one hand and relative sensitivities on the other is defunct. I feel like the Duke of York who marched you up a hill. I have to march you down again, but I hope we shall finish up on the other side, as indeed the original Duke of York must have done.

During the last few years it has been fashionable to study visual pigments in the living eye. The relevant principle was used qualitatively by Abelsdorff (1897) and by Hosoya (1929) many decades ago, and was first put on a quantitative basis in the early fifties simultaneously by Brindley and Willmer (1952) and by ourselves (Weale, 1951, 1953). I like to refer to the technique as dowsing because it is used largely for divining rods. The principle is simple (Fig. 9). When the eye is fully dark-adapted and light-



Reproduced by permission.

Fig. 9. The principle underlying fundus reflectometry (Weale, 1953).

absorbing substances have accumulated in the outer limbs of the rods and cones, the fundus appears a little darker than when the eye has been exposed to a bright light and the pigments have been bleached. The maximum change observable in man is about 16 per cent. This is small, but measurable.

In order to be able to express this change in the apparent lightness of the fundus in terms of vision, it is advisable to use, not white light as in an ophthalmoscope, but filtered or monochromatic light. We find, in general, that the intensity of such a beam of light, which traverses the retina twice—once before and once after reflection at the fundus—emerges weaker from the eye during dark than during light adaptation. There is, of course, no need to restrict our measurements to only one type of coloured light: many an interesting fish has escaped through the spectrometric mesh when the gaps were left too wide. In the method which I have been using during the last four years, twenty-six spectrum lights are used in very rapid

Reproduced by permission.

Fig. 10. See text (Weale, 1959).

succession. In fact, the fundus is scanned by the whole spectrum in less than a second. On emerging from the eye under test, these rays are collected by a photo-cell. The resulting photo-current is greatly amplified and displayed on a cathode-ray screen when it is photographed and produces a characteristic trace (Fig. 10). This shows a number of peaks which occur essentially in pairs. Every pair is produced by one of the colours of the scanning spectrum. The more light emerges from the eye the higher do the peaks rise. The fact that there are two peaks is due to an arrangement which provides a control for the constancy of the light-source, the photo-cell and the cathode-ray oscillograph. Thus the right peak in each pair is due to light reflected from the human fundus, the left one due to the control. The provision of suitable fixation marks enables one to explore any part of the eye. Now we know from the characteristics of visual purple, the pigment found in human rods, that when intact it impedes the transmission largely of blue-green light. In other words, it preferentially absorbs such light, and this is why our rod or night vision is particularly sensitive to blue-green. *A priori*, therefore, one would not expect a uniform change in the transmissivity of the retina as measured when the

living eye changes from the dark to the light-adapted state. We see that this expectation is fulfilled by superposing traces we have obtained on one and the same eye first in the dark-adapted, then in the light-adapted state. In the periphery this change occurs largely in the blue-green part of the spectrum, in the fovea it centres in the yellow green (Fig. 11). This slide shows you a manifestation of the physical basis of the Purkinje shift, recorded for the first time in one and the same human eye.

Quantitative results, culled from these and other data, are shown in Figure 12. First let us concentrate on the foveal data. The curve is

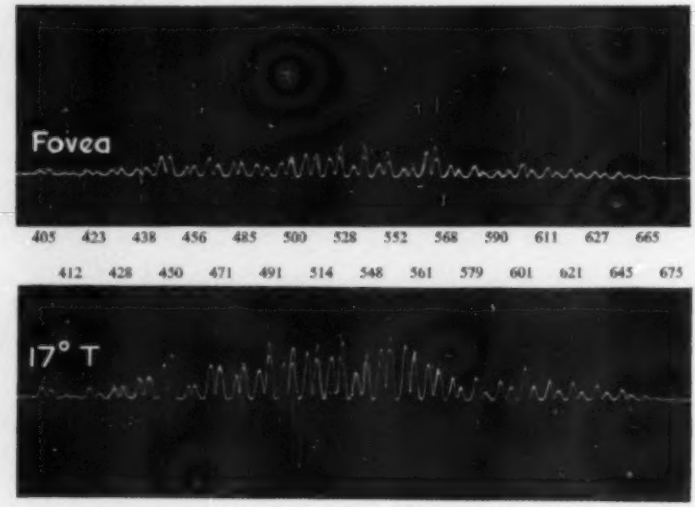


Fig. 11. Superposition of traces for the light- and dark-adapted eye, relating to the fovea and temporal retina respectively. (The wavelengths of the measuring lights are shown between the two traces.)

double humped. This can be shown to be due to the presence of two independent substances, one absorbing maximally in the green at a wavelength of about 540 mμ, the other at about 600 mμ. The former is the first pigment to be discovered in a rod-free area (Rushton, 1955), and was first found in the squirrel retina (Weale, 1955) (Fig. 13). It might therefore be called primochrome. The other is secundochrome, the second pigment. If we obtain good evidence for a third, it can be called tertiochrome. These names have two important advantages over the terms " chlorolabe " and " erythrolabe ", in that they assonate less with the labour of the studies and remind me less of my pitiful ignorance of Greek.

You will notice that although, on the basis of the Young-Helmholtz theory, one would expect the fovea to contain three types of pigment, there is no conclusive evidence of any change in absorption in the blue part of

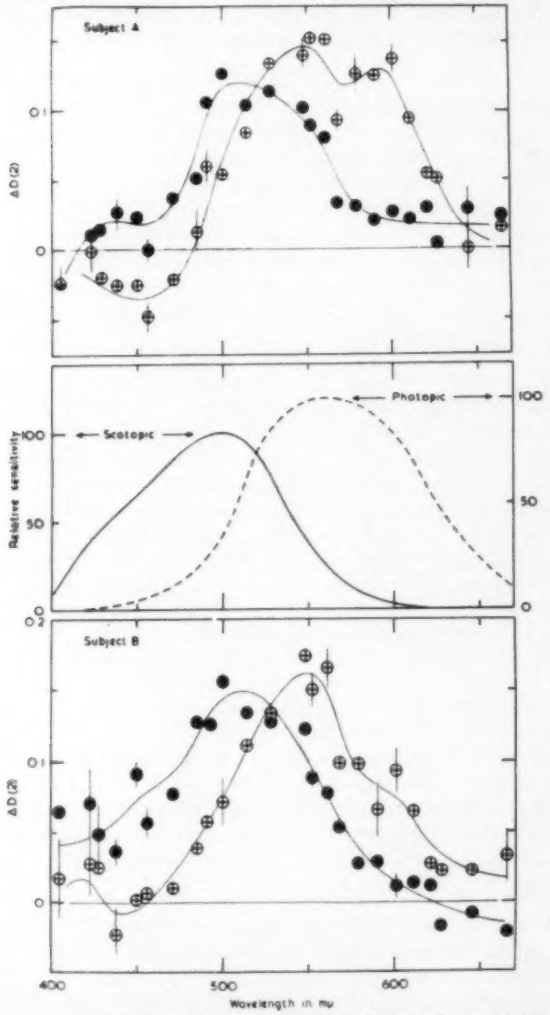


Fig. 12. Comparison between human scotopic and photopic sensitivities on the one hand (centre) and the changes in concentrations of light-sensitive substance on the fovea and periphery respectively, on the other. ⊕ = fovea; ● = 13° (A), 10° (B) temp. retina.

the spectrum. In my opinion this neither invalidates the Young-Helmholtz theory nor does it support the view that the whole of the fovea is tritanopic (or blue-defective). At best it reflects on the small amount of blue-sensitive pigment present in the fovea, at worst it may mean that the technique breaks down in this spectral region.

The peripheral data, which, like the foveal ones, are in broad agreement with Rushton's fine work, show good evidence of the presence of changes due to visual purple bleaching. They seem to be complicated, however, by features perhaps caused by secondary reactions. Experiments in progress at the present time may help to decide whether the short-wave hump is due to a separate blue-sensitive pigment, namely tertiochrome, or to some other factor. On the available evidence, we would expect tertiochrome to be present in larger amounts in peripheral than in foveal

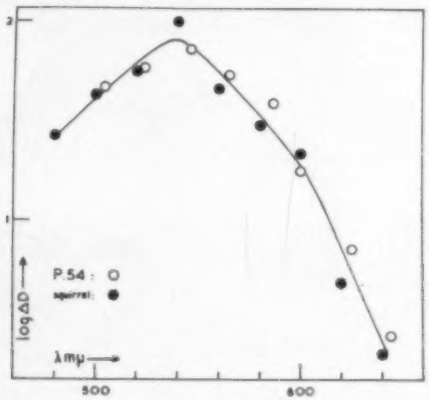


Fig. 13.

cones. Other evidence suggests that even these peripheral data can be contaminated by changes due to the two main cone-pigments, primo- and secundochrome, which may also be present in peripheral cones. It is a moot point whether primo- and secundochrome may not perhaps be changed to visual purple when the retina is destroyed, and the pigments are extracted.

We now come to the point where, as I promised, I lead you down the hill again. The maximum change in the pigment density produced by bleaching provides an estimate of the concentration of the pigment in the rod or cone. Now we see that the density changes as measured in the fovea and periphery are of the same order. There certainly is no factor of 100 or 1,000 favouring the rods over the cones. If anything, the cones are slightly better off than the rods, a point I would not care to argue on the

strength of these raw results. What I wish to emphasize is that just as the thresholds of single rods and cones are of the same order, so are the effective concentrations of rod and cone pigments of the same order. Thus while the final threshold situations may be explicable in terms of pigment concentrations, the rate of dark adaptation must be largely a neural matter.

So much for my introduction. Where does the Duplicity Theory lead from here? Let me recall the stalwarts' view, namely that rods operate our night or scotopic vision and cones our daylight or photopic vision. I hope to have shown you how this statement depends on what we are viewing. It is true of the tree silhouetted against the night sky, but not of the stars which make the silhouette possible. We should not allow ourselves to be brow-beaten by a theory to the extent of designing our experiments on the assumption that it cannot be wrong. A more fruitful hypothesis would be that there has never yet been a theory which graced its beckoning finger with an eternity ring. Where, then, are our needs?

To begin with structure, we need more data on the relative number and distribution of rods and cones in normal human retinae. We do not possess a single datum on retinae obtained from colour-defective eyes with the celebrated and dubious exception of Larsen. Sjøstrand is wielding the electron microscope with cunning and success, yet—we have little idea about the initiation of the visual impulse in the receptor, and none about any possible differences between rods and cones. Noell has shown in brilliant experiments that cones are not poisoned by doses of iodo-acetate which are fatal to rods. Again, this finding will be understood only when the chemistry of the poisoning is cleared up. I admit, however, that Noell's discovery is one which stalwarts will cherish for a long time to come.

Another point requiring urgent attention is the light reflex. You remember that the optic nerve fibres which mediate pupillary constriction fail to relay in the lateral geniculate body. The response of the pupil differs accordingly as we stimulate retinal areas rich in rods or cones. Data for visual acuity lead one to deny that special receptors deal with this reflex. Is the operation of the light reflex then another separate example of the duplicity in post-receptoral organization?

Then take the electro-retinogram. There can be few topics in this subject in which extravagant claims have been based on flimsier evidence. I for one have yet to be convinced that a number of effects ascribed to rods or to cones may not be simply due to both or the other: and are, in a sense, artefacts due to an insufficiently intelligent control of stimulus conditions. The confusion reigning in this sphere is ably documented by Dodt and Walther, who found in the electro-retinograms of pure rod animals characteristics usually associated with cone activity. To

quote from a different field, it is often stated that visual acuity and sensitivity are basically antagonistic—good visual acuity being mediated by cones and high sensitivity by rods. The logic of this statement is astonishing: for visual acuity is measured with strong, but high sensitivity with low stimulus intensities. When the conditions are more truly comparable, acuity and sensitivity run in a sense parallel, for resolution holds its elder brother perception firmly by the hand.

Again, can rods fulfil any function in daylight vision? Aguilar and Stiles have shown that rods can mediate intensity discrimination at levels high as compared with the illumination now prevailing in this hall. You may well ask whether the visual pigments do not get bleached. Now one finds that visual purple in the living human eye begins to bleach measurably only at luminances corresponding to a bright summer sky. Furthermore, you can dilate one of your pupils with a mydriatic and then step into the summer. If you close each eye in turn, you will notice, I think, that the eye with the dilated pupil is more sensitive to red and less sensitive to blue than the normal eye. This suggests an extension of the Purkinje shift at photopic intensity levels. I conclude tentatively that, under normal conditions, we do not really experience rod-free vision and that truly photopic vision of large visual fields might, in the long run, be intolerable.

However, I do not wish to blur unduly such distinctions as help us rationalize our ideas. I think the embryologist is called upon to make a great contribution to sensory physiology in general and to our theory in particular. How is it brought about that receptors, such as cones, are associated with elusive photo-chemical substances and a specialized neural system, while the rods with their visual purple go for integration? When are these pigments formed in the living duplex eye? Which, if any, parts of the retina develop functionally only as a result of exposure to light? We have some evidence from frogs, rats, and also kittens which are born blind. What about man? Do the pigment precursors act as organizers or is it a neural transmitting or inhibiting substance which plays this role? There, one feels, lies the crux of much duplicity.

But to conclude let us remember that, though the theory may still be vague, its practice is clear. Well may we wonder why the cone-system appears to adapt much faster than the rod system; we always wonder why. With the cones we dart from light to shade and back again, and rapid adaptation is something which a nimble brain needs to survive. What of the rods? If our fathers decide to blast an atomic trail back to the life of cavemanship where man rests in the light and hunts in the dusk, then our children will be vouchsafed a consoling thought: the thought, namely, that the speed with which our rods adapt to failing light is adequate for the rate at which the sky loses the rays of the setting sun.

REFERENCES

ABELSDORFF, G. (1897) *Z. Psychol. Physiol. Sinnesorg.* **14,** 77.
AGUILAR, M., and STILES, W. S. (1954) *Optica Acta.* **1,** 59.
ARDEN, G. B., and WEALE, R. A. (1954) *J. Physiol. (Lond.)* **125,** 417.
BAUMGARDT, E. (1949a) *Rev. Opt. (theo. instrum.)* **28,** 661.
——— (1949b) *C. R. Soc. Biol. (Paris)* **143,** 786.
BERGER, C., and SEGAL, J. (1950) *C. R. Soc. Biol. (Paris)* **144,** 478.
BRINDLEY, G. S., and WILLMER, E. N. (1952) *J. Physiol. (Lond.)* **116,** 8.
DARTNALL, H. J. A. (1948) *Brit. J. Ophthal.* **32,** 7.
——— (1960) *Nature* (in press).
DODT, E., and WALTHER, J. B. (1959) *Pflüg. Arch. ges. Physiol.* **268,** 204.
EDRIDGE-GREEN, F. W. (1920) *The physiology of vision.* London, G. Bell.
GRANIT, R. (1947) *Sensory mechanisms of the retina.* Oxford Univ. Press.
HOSOYA, Y. (1929) *Tôhoku J. exp. Med.* **12,** 146.
NOELL, W. K. (1959) *Amer. J. Ophthal.* **48,** 347.
PARSONS, J. H. (1924) *Colour vision.* Cambridge Univ. Press.
RUSHTON, W. A. H. (1955) *Farbe.* **4,** 219.
———(1958) *Symposium on the visual problems of colour.* Teddington, National Physical Laboratory. H.M.S.O.
SJØSTRAND, F. (1959) *Ergebn. Biol.* **21,** 128.
STILES, W. S., and CRAWFORD, B. H. (1937) *Proc. Roy. Soc. B.* **122,** 255.
WALD, G. (1954) *Science.* **119,** 887.
——— (1958) *Symposium on the visual problems of colour.* Teddington, National Physical Laboratory. H.M.S.O.
WEALE, R. A. (1951) *Inst. Ophthal. Ann. Rep.* **3,** 18.
——— (1953) *J. Physiol. (Lond.)* **122,** 332.
——— (1955) *J. Physiol. (Lond.)* **127,** 587.
——— (1958) *Nature* **181,** 154.
——— (1959) *Optica Acta.* **6,** 158.

FACULTY OF ANAESTHETISTS

AT A MEETING of the Board of Faculty of Anaesthetists on 21st December, Dr. Geoffrey Organe was elected Joseph Clover Lecturer for 1962. Nuffield Prizes were awarded to Dr. Margaret Annie Branthwaite (University of Cambridge and University College Hospital) and to Dr. Arumugam Ganendram (University of Bristol).

BERNARD JOHNSON MEMORIAL

THE DEAN OF the Faculty of Anaesthetists wishes to announce that the Bernard Johnson Memorial Fund will be closed at the end of March 1961. An anonymous donor has generously agreed to double any contributions received between 1st January and 27th March 1961 up to a maximum of £250. Donations may be addressed to the Dean of the Faculty at the College.

NATURAL HISTORY OF ARTERIOSCLEROSIS OF THE LOWER EXTREMITIES

Hunterian Lecture delivered at the Royal College of Surgeons of England

on

22nd April 1960

by

K. Bloor, M.D., F.R.C.S.

Manchester Royal Infirmary

INFORMATION ABOUT THE course of arteriosclerosis has been imprecise or entirely lacking in the past. In the face of the clinical tragedies of myocardial infarction and limb amputation, the clinician's helplessness has led him to take a more gloomy view in individual patients than is warranted by the facts and in this he is supported by most written opinions. For example, Allen, Barker and Hines (1955) have said that 50 per cent. of patients are dead in three years.

In 1947 Professor A. M. Boyd set up a follow-up clinic at the Manchester Royal Infirmary, with three aims, firstly to find out what happened to the life and limb of the patient with arteriosclerosis, secondly to provide a standard of comparison for the variety of treatments which could be expected to arise, and thirdly to investigate the cause of the disease. This lecture is mainly concerned with the first two of these aims, the establishment of facts, and the consideration of present day practice in their light.

To do this, it was necessary to see a large number of patients and to follow them for a long time. It was decided to follow up all those patients with arteriosclerosis who presented with intermittent claudication and had no pregangrene or gangrene. Because of our known interest in this disease, patients were referred to the clinic who were not suitable for surgery and it is felt that they are relatively unselected compared to other surgical series.

Between 1947 and June 1953, 1,508 patients of this kind were seen, and it was then decided that this was probably enough. 20 patients by reason of residence abroad or other factors could not be followed up. 12 of the remaining patients have been lost to follow-up, leaving 1,476 for consideration.

Diagnosis has been described before (Boyd *et al.*, 1949). Most of the younger patients have had arteriography, but it has not been done in the others unless the performance of an operation allowed it. If any uncertainty in diagnosis occurred the patient concerned was reviewed until events or investigation resolved it. If doubt has persisted the patient

has not been included in this series. At this first visit the anatomical site of the lesion was determined by clinical examination and oscillometry.

They were seen at three-monthly intervals or more frequently if necessary in the early stages of their disease, and later at six- and twelve-monthly intervals. As they died, the cause of death was obtained from the general practitioner or hospital. If the patient moved away or was unable to attend, then the help of a local clinic or his general practitioner was sought in doing an adequate follow-up. Patients have been surprisingly willing to travel long distances to attend the clinic.

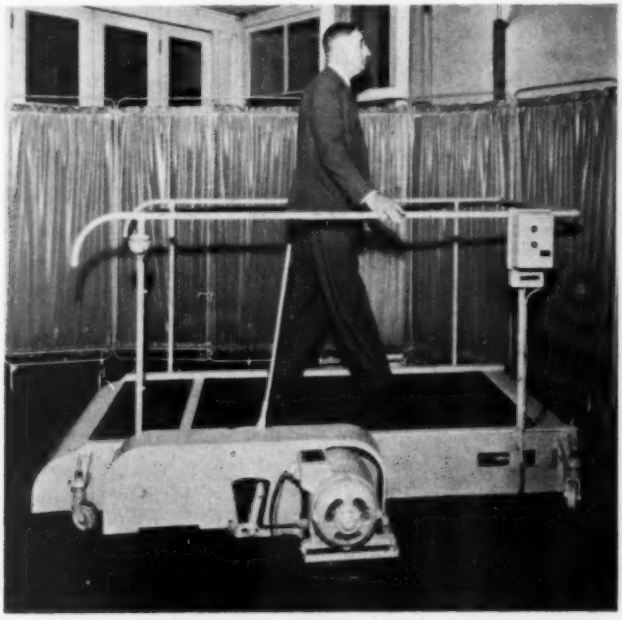


Fig. 1. Walking machine.

At his initial visit the patient's halting distance was determined on a machine designed to mimic normal walking (Fig. 1). It consists of a moving belt which slips over a platform and is driven by an electric motor through continuously variable gearing. The patient is put on the machine at a low speed and the speed is increased until it reaches what he considers to be his normal pace, which is then held. He is walked at this speed at each visit afterwards. At each visit a full limb examination was done and peripheral pulses and oscillations noted.

The patients have in the main been treated conservatively. None of them in the period of review had direct arterial surgery. The operations done were lumbar ganglionectomy, amputations and tenotomy of the tendo Achillis.

Age and sex incidence

Of the 1,476 patients, 156 (10.5 per cent.) were women and their average age at onset was 62.2 years contrasted to the male average age of 57.5 years. Table I shows the age of the patients at the onset of claudication.

TABLE I

Age	*Men*	*Women*
25—34	7	—
35—44	83	8
45—54	418	24
55—64	557	57
65—74	231	60
75+	24	7
TOTAL	1,320	156

Age at onset of intermittent claudication.

In view of the later age of onset and the comparative infrequency of the disease in women, it could be expected that their further history would be better than in men of the same age, but it is in fact very similar. It appears that once a woman has arteriosclerotic symptoms the feminine advantage is lost and she suffers just as badly as the men. When the figures for the women are broken down by age, the resulting groups are too small for generalization. In most of this lecture, therefore, the male patients only will be used for illustration.

Diabetes mellitus

Those with diabetes mellitus were a very small part of the total, only 4 per cent. In some of these the excretion of sugar in the urine started after the onset of claudication and therefore the proportion with diabetes in an overt form at first attendance was smaller than this. The death rate in diabetics appears to be only a little greater than in the other patients.

Review period

The 1,476 male and female patients have been followed-up in detail up to June 1957, and thus have been reviewed for four to 10 years if they survived. 660 of them were seen during the first year of symptoms, but many came only in the second or later years, and a few as long as 10 or more years after onset. As will be seen, the course of events in the first year differs from that in subsequent years, so that death rates and amputation rates have been calculated according to the year from the onset of intermittent claudication.

660 were seen in their first year after onset and of these 32 died. Another 289 were seen in their second year of symptoms, giving 917 under review in that year. This increases to 1,065 in the fourth year, after which the numbers decline as the period of follow-up of the survivors comes to an end.

When I refer to the year of onset, I am referring to the onset of claudication. Some patients, however, attend with angina pectoris or a coronary or cerebral thrombosis which may have predated the leg symptoms by many years. 7.9 per cent. of patients had some central arterial lesion prior to their intermittent claudication.

Death rates

Nearly all patients with arteriosclerosis can be expected to die of their arterial disease. Of the 673 deaths which occurred, 401 (59.5 per cent.) were cardiac, 112 (16.6 per cent.) cerebral and another 55 (8.1 per cent.) directly due to vascular disease, such as aneurysmal rupture, mesenteric occlusion and so on, and only 105 (15.6 per cent.) died of other intercurrent disease.

TABLE II

ESTIMATED PERCENTAGE SURVIVAL TO 5 AND 10 YEARS IN MEN.

Age	5 *years*	10 *years*
35—44	91.8	77.6
45—54	79.0	54.2
55—64	72.0	34.9
65—74	60.3	22.2
75+	40.3	0

Survival rates for the men have been estimated from the death rates observed in each year and are presented in Table II. In the age group 45–54 years, the five-year survival was 79 per cent., the 10-year survival 54 per cent. This expectation diminishes with age to a 10-year survival of 22 per cent. in the age group 65–74 years.

These figures are better than expected and raise the question of how much worse this is than in the general population. The survival rates of the general male populace of the South East Lancashire area have been estimated. In the age group 45–54 years this is 93.5 per cent. at five years, compared to 79 per cent. in the arteriosclerotics.

Figure 2 shows that the survival rates of the 55–65 age group in the arteriosclerotics roughly parallels that of the general population of 65–74 years and, as a rough approximation, arteriosclerotics can be thought of as being 10 years older than they should be.

Non-fatal coronary and cerebral disease

The damage done in central arteries does not end with these fatalities. Non-fatal coronary or cerebral disease may produce angina of effort or hemiparesis. This will occur in 20 per cent. of patients at least once if they survive to five years and possibly oftener.

At the time of these non-fatal coronary occlusions there are few coincidental occlusions of other vessels. Of 224 non-fatal attacks observed, two patients had a more or less coincidental occlusion of another artery occurring suddenly and five had one in the following month. This is rather curious as coincidental occlusions might be expected for two reasons.

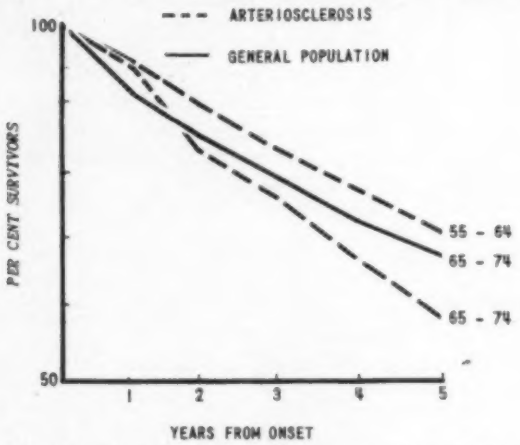


Fig. 2. Estimated survival rates of 10-year age groups of arteriosclerotics and general population.

The first is peripheral embolism. It is generally accepted that peripheral embolism is a highly fatal condition. Up to 50 per cent. of cases admitted to hospital which occur following coronary thrombosis die in hospital. But this is hardly high enough to explain the infrequency of peripheral embolism in these surviving patients and it is felt that embolism must be rather a rare condition following myocardial infarction. The second reason is theoretical. The theory is the general one, variants of which are always arising, which says that a block is produced by acute changes in some blood constituent, such as the lipids or the factors concerned in coagulation. If this is true, it should be so for the peripheral arteries as well, but these figures do not support such theories.

Prognosis in limbs

It is very noticeable in this series that although a few patients have upper limb vessel blocks, they seldom produce symptoms and no patient has

lost arm or finger due to gangrene. Patients do complain sometimes of mild Raynaud's phenomena, often in a finger which has been subjected to minor injury in the past, but this seldom lasts more than two or three years.

In the lower limb a variety of events can occur. A patient may arrive at the clinic with a segmental occlusion, for example, in the popliteal artery, which may not extend immediately, and his claudication may im-

Fig. 3. Diffuse arteriosclerosis.

prove with the development of his collateral circulation. Later on he may develop a further occlusion in any site in the same leg and again he may recover or he may progress to pregangrene, gangrene and amputation. Another patient may develop gangrene without obvious vascular deterioration.

To present all this calls for simplification, and to do this I have referred only to the first event of a kind in any patient in working out their rates of incidence. If, for example, a patient has an additional femoral and then

a common iliac block, only the first is included in working out rates of incidence, thus putting these a little on the low side, but both events are included in the gross figures.

The patients' lesions on being first seen were as follows:

36.1 per cent. had femoral thromboses, and nearly a quarter of these were bilateral.

32.0 per cent. were popliteal and only an eighth were bilateral.

4.2 per cent. had common iliac blocks and 1.6 per cent. aortic blocks.

25.1 per cent. were thought to have grossly narrowed arteries without a complete main vessel block (Fig. 3). This lesion is usually bilateral.

Bilateral lesions imply more severe disease and they have a higher death rate than unilateral lesions in the same age group. Oddly enough, they are more frequent in the younger patients. 64.7 per cent. of the 35–44s have bilateral lesions, compared to 38.1 per cent. in the age group 55–64 and 32.2 per cent. in the age group 75+ years. This is only one example of the poorer state of the limbs of the younger patient, in spite of his longer survival.

Further thrombosis

It is quite easy to diagnose further vessel occlusion by history, clinical examination and oscillometry, if the previous state is known. This is particularly so if the vessel occluded is the common iliac or aorta, as disappearance of the femoral pulse is easy to detect.

335 patients had an addition to their vascular occlusion during the period under review. Of 901 patients who presented with lesions in one leg, 163 later had a thrombus in the other leg, and 65 had further lesions on the side first complained of. In 367 patients with narrowing only, 54 subsequently blocked a major vessel. In 208 patients with bilateral blocks on attendance 53 had a further thrombus. The frequency with which this occurs is fairly large, and more or less the same in each age group. The surviving patient has a 25 per cent. chance of having increased the occlusion in his legs at the end of five years. The chance at 10 years is about 45 per cent. Those people who had a further main vessel block in a leg previously blocked numbered 118. In only 14 (11.9 per cent.) instances did this lead to midthigh amputation.

An interesting special case is that of blocks of the common iliac and aorta. These made a total of 5.8 per cent. when first seen, but 94 more developed common iliac blocks and 21 developed aortic blocks during observation, about another 5 per cent. The usual clinical course observed is for one common iliac to block followed then by extension to the aorta with blockage of the other side.

It might be expected that a common iliac thrombus above another main arterial block would inevitably lead to gangrene, but midthigh amputation was necessary in only 8.7 per cent. of cases. This is an indication of the excellence of the collateral circulation which can develop around the ilium and across the pelvis from the common iliac of the other side. It is also an indication of the frequency with which the common femoral artery remains open, preserving the flow through the profunda femoris. After extension to the aorta, however, the loss of collaterals is too great for the limb and 50 per cent. of patients lose a limb.

Narrowing of the common iliac artery occurs and an idea of its frequency can be obtained by listening to the femoral artery in the groin, when a systolic murmur can be heard in about 40 per cent. of cases. This murmur is variable and can be changed by varying the local conditions of blood flow in some way, such as by exercising the patient or warming the leg, so that it is not entirely a reliable and constant sign of narrowing but it can be a useful diagnostic sign in some obscure cases.

Pregangrene and gangrene

Gangrene is relatively uncommon after a second major vessel block in a limb. This shows the importance of blockage of the smaller peripheral vessels such as the tibials or the digital arteries, in determining the onset of tissue death and gangrene, and in these patients multiple or extensive blocks are shown on arteriography. Without an arteriogram, these distal blocks can only be inferred, but it would be a mistake to suppose that they must occur in every case. The onset of gangrene in a limb may not be determined solely by a decrease in the blood supply to a limb. Many limbs in a stable vascular state are lost by the carelessness of the patient whilst cutting his toe nails or by his doctor in prescribing unnecessary local treatment. Minor trauma plays a large part in precipitating gangrene.

Gangrene and pregangrene are loose terms difficult to define. Gangrene usually implies the death of a volume of tissue, such as a toe or a forefoot. Since it is not possible to predict the course of minor ulceration or rest pain, any episode of rest pain and of breach of the skin accompanied by rest pain are here classified as pregangrene which may lead to gangrene and amputation.

304 episodes of pregangrene and gangrene were seen, involving 263 patients, as 41 were bilateral.

Midthigh amputation was done 142 times, bilaterally in 21 cases, and therefore 121 patients were affected.

This is only one fifth the number of those who died. Ischaemia is a less pressing problem than death at present in these patients.

The number of ischaemic incidents varied with age (Table III), and in this the younger patients were again slightly worse off, with an average incidence in the first five years of 4.6 per cent., compared to 3.5 per cent. and 3.6 per cent. in the 45–54 and 55–64 age groups.

The number of times this led to major amputation was also proportionately greater in the younger patients, 38.4 per cent. in the 35–44 age group falling to 23 per cent. in the 45–54 age group before rising again.

Pregangrene and gangrene are commoner in the first year after onset. They were seen in 8.3 per cent. but led to amputation in only about a quarter of these. In the third year the ischaemic rate is 3.0 per cent. and about half of these are amputated, meaning that the amputation rate in each year is not greatly different. The greater incidence and greater recovery are due to the fact that the collateral circulation in the first year has not yet fully opened up and therefore the feet are less protected, but the ability to retrieve minor degrees of gangrene is also greater.

TABLE III

Age	*Pregangrene and gangrene average percentage per year in the first 5 years*	*Percentage with pregangrene and gangrene proceeding to midthigh amputation*
35—44	4.6	38.4
45—54	3.5	23.3
55—64	3.6	42.0
65—74	6.6	41.0
75+	8.4	57.0

Age and gangrene.

The rate at which midthigh amputation was done was much less than expected and was for all men an average of 1.4 per cent. in the first five years. A man surviving five years has a 7 per cent. chance of losing a leg, and if he survives to 10 years, a 12 per cent. chance.

The amputation incidence does not vary much according to the lesion found at the patient's first attendance except in one instance. This is in patients with common iliac or aortic blocks. Their amputation rate as the years pass is two and a half times greater than in the others.

Intermittent claudication

Some patients achieve a surprising degree of relief from intermittent claudication. The most astonishing seen was in a man who had an aortic thrombosis to the level of the renal vessels, demonstrated by aortography. About 18 months after he was seen he improved enough to be able to walk half a mile without any pain.

To discover the degree of recovery possible in non-diseased arteries, patients with arteries blocked by trauma, surgical or otherwise, who were

seen shortly after injury and were thus relatively unselected, were reviewed. They were treated, if at all, by lumbar ganglionectomy. The arteries blocked were five popliteals, four femorals and three external iliacs, 12 in all, in people with an average age of 26. The only middle-aged patient, who was 49 years old, did not improve. Two improved very considerably and the other nine could walk half a mile without pain. One has recently taken up cross-country running, but his degree of success at this is not yet known. The time taken for major recovery was from six to nine months, but one patient seen the day after injury already had no

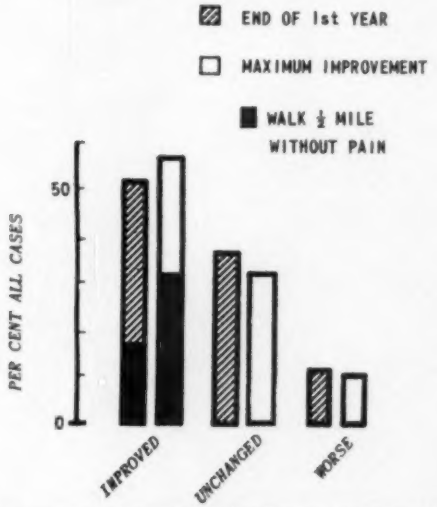


Fig. 4. Improvement in intermittent claudication, age group 45–64 years.

intermittent claudication despite division of the femoral artery. It is obvious that the leg with an undiseased collateral bed may return almost to normal function, and does so fairly quickly.

In any arteriosclerotic patient the relative amount of recovery observed will depend on the degree to which the collateral circulation was developed before he is seen, and also on whether his onset was sudden or gradual.

If the occlusion is gradual then he may begin to develop his collaterals well before symptoms appear and has less room for improvement once they do start. If a patient gives a history of sudden onset of symptoms he is more than twice as likely to improve as the patient with a gradual onset, which it is well to remember when giving a prognosis. Improvement in these patients has been seen for as long as three years after the onset.

The patients in the age range 45–64 who were seen in the first year of symptoms have been taken to illustrate the improvement to be expected. There were 363 of them, and 18 could already walk half a mile without pain when they were first seen. They were divided up according to the severity of symptoms when first seen, as it was expected that the ones who halted at a very short distance initially would prove to recover the least. It was, however, found that recovery was very similar whatever the initial distance.

Fig. 5. Narrowing of branches of femoral artery.

Many of these did eventually have a further thrombus and therefore got worse, but their recovery up to that point was considerable.

39 (10.7 per cent.) were worse without apparent cause; 118 (32.5 per cent.) were unchanged and 206 (56.7 per cent.) improved. Improvement is defined as showing a consistent improvement of more than 100 yards in halting distance, but many did considerably better than this. 114 or 31.4 per cent. (including those 18 able to do so when first seen) were able to walk more than half a mile without halting. In this group the improvement has been maintained on average for 4.5 years until they died, had a further thrombus or ran out of follow-up.

A great deal of this improvement occurs after the first year. The state at the end of the first year compared to that ultimately achieved is seen in Figure 4. Although there is no difference in the unchanged and worse groups, the number of patients able to walk more than half a mile without pain has doubled.

This recovery is not as good as that achieved by non-diseased arteries. In part the reason for this is the habit of atheroma of gathering at the mouths of small vessels. Since these patches cannot dilate, the size of the collaterals which these vessels can form is limited at their origin. This can be seen in almost any arteriogram, as shown in Figure 5. Two of the branches of the femoral artery are so narrowed at their origin as to look completely blocked.

The prognosis for the limb can be summarized at this point. At the end of five years survivors in the 55–64 age group have a 25 per cent. chance of having a further clinically detectable thrombosis which may lead to midthigh amputation in 3 per cent. Another 4 per cent. will also have a midthigh amputation, leaving 71 per cent. From the figures just given, 56.7 per cent. of these will have improved their intermittent claudication, that is 40.3 per cent. of the whole, and roughly half of these will be able to walk half a mile without pain. It could also be put the other way round, that 59.7 per cent. will be worse or unchanged.

Lumbar ganglionectomy

To assess the influence of a single act such as lumbar ganglionectomy when superimposed upon the fluctuations of this disease is a difficult task. Any patient may experience a course which if expressed graphically would resemble the ups and downs of a scenic railway, ending at a lower level than the starting point, but it does not have the predictability of this physical model. To estimate the effect of lumbar ganglionectomy therefore demands a large control group, but comparison may still be inaccurate, as it is always possible that the patients operated on may have been worse or not improving as much as expected, and not strictly comparable with the others.

Opinion has differed on the effectiveness of lumbar ganglionectomy (Telford and Simmons, 1946; Mavor, 1955; De Takats, 1958), and the most persuasive in recent years in its favour is by Smithwick (1957). The results from the Vascular Clinic at Manchester Royal Infirmary were recently given in a review (Boyd and Bloor, 1959), but no attempt was made to give a comparison with controls.

278 patients in the age group 45–64 years had lumbar ganglionectomy in the review period. 273 were bilateral; this was done even when the disease appeared to be predominantly unilateral. There were two post-operative deaths, a mortality of 0.7 per cent. In addition there were six

post-operative extensions of thrombus in the legs, four coronary thromboses and two cerebral thromboses, an arterial morbidity of 4.3 per cent. The subsequent death rate of these patients is lower than that in patients not operated upon, which goes to demonstrate our success in choosing the fitter patients for operation.

The results will vary with the time at which it was done and the motive for doing it.

When done in patients who were getting worse, no improvement was seen in any and some continued to get worse.

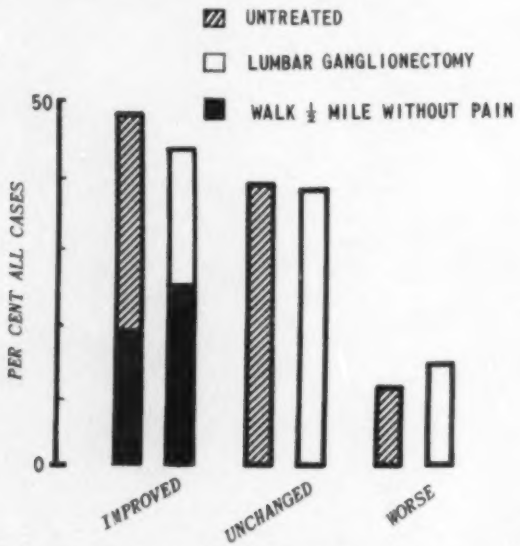


Fig. 6. Maximum improvement after lumbar ganglionectomy compared with those not operated upon, age group 45–64 years.

74 patients were operated on more than a year after onset. They may have been fairly static and were certainly not getting worse at the time of operation. It would be difficult to find exactly matched controls for these, for age and stage and date in the disease, and it is felt that a large element of bias would certainly appear during selection if attempted.

23 improved and 10 could ultimately walk more than half a mile without pain. Nine were worse. Thus 31 per cent. improved and 69 per cent. were unchanged or worse. These figures are not very impressive, either as a return for effort on the part of the surgeon and the patients, or as an indication of the therapeutic efficiency of lumbar ganglionectomy.

The last group considered contains the patients aged 45–64 years, operated upon in their first year of symptoms. They were done towards the end of the first year, nearly all in the last three months. They are a selected group, worse than the general run of patients, as is obvious when looking at their walking distance at the end of the first year. None can walk more than half a mile without pain. They can be compared with the patients in the same age group who were unable to walk more than half a mile without pain at the end of the first year.

42 patients had lumbar ganglionectomy and the controls numbered 262 (Fig. 6). 48.2 per cent. of the non-sympathectomized improved and 45.1 per cent. of those operated on. The proportion able to walk half a mile without pain was 26.1 per cent. and 18 per cent. respectively. Those sympathectomized were slightly younger on average and were generally fitter than the others. They should therefore have had better results. It is felt, therefore, that lumbar ganglionectomy has not been shown to improve claudication.

Lumbar ganglionectomy and further thrombosis

It has been stated by Smithwick (1957) that one of the great benefits of the operation is that it prevents the development of further occlusion in the legs. Since nearly all the sympathectomies were bilateral comparison is possible with events in both legs in those not operated on. 65 of 278 patients operated upon had a further occlusion after operation. When this is reduced to a rate per year the incidence in either leg is 4.4 per cent. per year in the first five years after onset, compared with 5.1 per cent. in the non-sympathectomized. Since again the better general condition of the patients operated upon must be remembered, there is no difference in the results, and no advantage in sympathectomy.

Lumbar ganglionectomy and gangrene

Lastly, is lumbar ganglionectomy worth doing for pregangrene and gangrene? It must be understood that this has not been done in this series for patients with massive gangrene, but for those with pregangrene as defined above and those, difficult to define, with what can be called minor gangrene, that is with a small amount of ulceration or necrosis combined with a fairly good blood supply. In this group 34 per cent. came to midthigh amputation shortly afterwards.

In the 45–64 age group those with pregangrene and gangrene not treated by sympathectomy had an incidence of midthigh amputation of 35.2 per cent. Whilst these number amongst them the milder cases, they will also include those to have progressed too far to recovery by ganglionectomy.

There is thus virtually no difference between them, and it is difficult to claim any improvement from ganglionectomy.

It would be idle to suggest that lumbar ganglionectomy produces no clinical effect. It is common experience that lumbar ganglionectomy does immediately improve some minor ulceration and gangrene, but it is doubtful whether even in these circumstances it can ever be said to have saved a limb. In the past lumbar ganglionectomy has certainly been done too frequently. It has been argued that the warm feet it gives are sufficient benefit in themselves, and certainly many patients say so, but a combined mortality and morbidity of 5 per cent. is surely too high a price to pay for this effect. The use of lumbar ganglionectomy in this condition should be restricted to the rapid alleviation of pregangrenous symptoms.

Arterial surgery

Evaluation of the results of arterial surgery must await more detailed reports of follow-up but some generalizations can be made now. Consider the surgeon with 100 patients aged 55–64 seen in their first year whose intermittent claudication he has successfully treated, the method used being at the moment immaterial. In the next five years 28 will die and about 20 will have a coronary or cerebral thrombosis, leaving 57 hale and hearty. 25 more will have a further thrombus meaning another operation or a recurrence of intermittent claudication. There will be at the end of five years 42 patients with unsullied benefit. If the same sum is done for the 45–54 age group, at this point there remains 48 patients, not very different from the above, and for the 34–44 age group 63.

Now to consider the results of the operations themselves. At present it would appear that the failure rate for artery grafts to the femoral and popliteal arteries is 50 per cent. or more in the first five years (Szilagyi *et al.*, 1956; Luke, 1958; Wylie and Gardener, 1955). In fact if it is taken as 50 per cent. this is being overkind to the operation. This will leave the surgeon with roughly 21 people still benefiting at the end of five years.

For aortic and common iliac lesions the picture is different. With a high immediate success rate and a failure rate which may be as low as 20 per cent. one would have 35 happy patients at the end of 5 years. When the additional risks of gangrene to these patients are considered, it is felt that all patients with a high arterial block should have direct surgery unless they have minimal symptoms. Surgery to arteries below the inguinal ligament is not worth while with present techniques if it is being done solely for intermittent claudication, unless this is causing considerable social embarrassment. Even in a young man a tenotomy of the tendo Achillis would be preferred if indicated (Boyd and Bloor, 1960) rather than an artery graft.

On the use of direct surgery to save limbs from amputation, little can be said at present. Published results are few and undetailed. Since these operations will usually be done for multiple blocks, relief of intermittent

claudication may not be a by-product of a successful operation. The high spontaneous recovery rate of pregangrene must also be remembered, particularly in the first year when it is 75 per cent. There is no reason for the surgeon to suppose that if he has successfully grafted a pregangrenous leg he has saved a limb, and the further survival of the graft may be doubtful.

It is to be hoped that technical achievements will allow the surgeon to extend his influence on this disease. More arteries, such as the renal and internal carotids, are coming to be of interest. However, the outstanding problem is that of death from coronary occlusion, and it is hoped that the future lecturer on this subject will be as much concerned with this as with the limbs, always remembering that if patients' lives are saved there will be many more limbs also needing preservation.

Summary

1,476 patients presenting with intermittent claudication due to arteriosclerosis have been followed up for from four to 10 years.

Death rates, the incidence of non-fatal coronary and cerebral disease, of gangrene and amputation are given.

Mortality and amputation rates are less than was expected.

Further limb vessel occlusions occurred in 20 per cent. of survivors in the first five years after the onset.

Common iliac and aortic occlusions carried a worse prognosis for amputation than other lesions.

Intermittent claudication, before further occlusion occurred, improved considerably in 59.6 per cent. and some improvement was seen up to three years after onset.

Lumbar ganglionectomy was not found to influence intermittent claudication, the rate of further thrombosis or the incidence of amputation in pregangrene and gangrene.

In considering the results of surgery, the central effects of the disease overshadow the peripheral ones and favourable five-year results are small because of this. It is suggested that arterial surgery is only generally applicable to the aorta and common iliac vessels at present.

ACKNOWLEDGMENTS

This study was initiated by Professor A. M. Boyd and other members of the Department of Surgery, and has been largely my responsibility since 1950. I owe much to the support and encouragement of Professor Boyd. The Medical Illustration Department of Manchester Royal Infirmary gave great help in presentation.

REFERENCES

ALLEN, E. V., BARKER, N. W., and HINES, E. A. (1955) *Peripheral vascular disease*. Philadelphia, W. B. Saunders.
BOYD, A. M., RATCLIFFE, A. H., JEPSON, R. P., and JAMES, G. W. H. (1949) *J. Bone jt. Surg.* **31B**, 325.
———— and BLOOR, K. (1959) In *Recent advances in surgery*. London, J. & A. Churchill Ltd.
———— (1960) *Brit. med. J.* **1**, 548.
DE TAKATS, G. (1958) *Arch. Surg. (Chicago)*, **77**, 655.
LUKE, J. C. (1958) Discussion. *Arch. Surg. (Chicago)* **76**, 260.
MAVOR, G. E. (1955) *Lancet* **2**, 974.
SMITHWICK, R. H. (1957) *Surgery* **42**, 415 and 567.
SZILAGYI, D. E., WHITCOMB, J. G., and SMITH, R. F. (1956) *Ann. Surg.* **144**, 611.
TELFORD, E. D., and SIMMONS, H. T. (1946) *Brit. med. J.* **1**, 386.
WYLIE, E. J., and GARDENER, R. (1955) *Surgery* **37**, 415.

ADMISSION TO THE COURT OF PATRONS

AT THE MEETING of the Council on 8th December, Lord Kindersley, the Chairman of the Appeal Committee, was admitted to the Court of Patrons.

A citation in honour of Lord Kindersley was given by Professor Sir James Paterson Ross, Bt., who spoke thus:

" Mr. President: It would be out of place as it is unnecessary for me formally to introduce Lord Kindersley to this Council for he is already a well-known and highly valued member of our community. He is, however, especially welcome today because we are all anxious to give expression to our feelings of appreciation and gratitude for the notable part he is playing in the life and activity of the College.

" It is only when we are called upon to serve on Hospital Governing bodies and Committees of Management that we become aware of the importance of the part played by laymen on these bodies; and the same applies in this College to the debt we owe to our lay friends for their help on the Finance and Appeal Committees and on the Committee of Management of the Institute of Basic Medical Sciences. Among these friends Lord Kindersley is *primus inter pares*, and those of us who have seen him in action can testify to the great benefits we derive from his shrewd judgment, his wise counsel and his inspiring leadership. For my own part I will never forget the comfort which his advice and support gave me when my actions were being criticised in the lay and medical press.

" Like most of the laymen who help us in our professional affairs Lord Kindersley is fully occupied already and it is hard to understand how he can find the time to work for us in the College at our meetings, and especially outside the College by influencing people on our behalf. His many activities in the City are in fact of great value to us because he has come to know personally so many influential people whom he has kindly approached in our interest—but that has meant hours and hours of solid work for which we are deeply grateful.

" It has been a privilege to become acquainted also with Lord Kindersley's family and those of us who were present on that very special occasion when he was admitted to the Honorary Fellowship will never forget meeting that wonderful old lady, his mother. This intimate note reminds us that it was Archie McIndoe who introduced Lord Kindersley to the College.

" It must be clear to everyone that Lord Kindersley's personal substantial support and continuing interest in our affairs entitle him to membership of the Court of Patrons and it is my privilege, Mr. President, on behalf of the Council to ask you now to admit him."

Fig. 1. The President admitting Lord Kindersley to the Court of Patrons, with Sir Stanford Cade (Vice-President) watching.

After being admitted to the Court of Patrons by the President, Sir Arthur Porritt (Fig. 1), Lord Kindersley spoke as follows:

" Mr. President, Sir James, Members of Council: This is a very proud moment for me; it is one which I shall certainly never forget, and I say in very real sincerity that I would like to thank you for the signal honour of making me a Patron of this great College. In the past few years I have developed, if I may put it that way, a very great respect for the Royal College of Surgeons and a very real affection for all those concerned with the College whom I have had the privilege to meet and be associated with. I am afraid that I do not really deserve this honour, nor do I deserve, Sir James, the very nice things you have said about me. It is my sincere hope that in the years to come I will be able to make some real contribution to the well-being of this great College and perhaps to show that the labourer is worthy of his hire."

APPOINTMENT OF FELLOWS AND MEMBERS TO CONSULTANT POSTS

J. K. A. CLEZY, F.R.C.S.	Specialist Medical Officer (Surgeon) in the Public Health Department of Papua and New Guinea.
G. L. GRIGG, F.R.C.S.	Honorary Thoracic Surgeon, Prince Henry's Hospital, Melbourne, and Assistant Thoracic Surgeon, Royal Melbourne Hospital.
R. L. B. BEARE, F.R.C.S.	Consultant Plastic Surgeon, St. Mary's Hospital, London, and Queen Victoria Hospital, East Grinstead.

PROFESSOR RAFFAELE BASTIANELLI

Senior Honorary F.R.C.S.

THE HIGHEST PROFESSIONAL distinction which the Royal College of Surgeons can confer is its Honorary Fellowship. 1913 was a vintage year which added to the Roll of Honorary Fellows the names of Raffaele Bastianelli, G. Crile, von Eiselsberg, Hartman, Will Mayo, J. B. Murphy and others. One only of this international galaxy of the surgical talent of half a century ago is still among us—Bastianelli—and it was the privilege of some of the Surgical Travellers to dine with this veteran of surgery, the senior Honorary F.R.C.S., in his home in Rome recently.

Fig. 1. Professor Raffaele Bastianelli

Professor and Signora Bastianelli, his American-born wife, live in a house which combines the grandeur of a Roman nobleman's villa with the interests of a private art gallery. It was here, surrounded by sculpture, paintings and works of art, that they provided their guests with food, wines, conversation and a warmth of welcome which seemed in their harmonizing perfection to belong to a bygone generation.

At the age of 96, Bastianelli is more alert in mind, quicker in repartee and better acquainted with world affairs than most men half his age. To some of us his recollections of Moynihan, Kocher, von Eiselsberg, Tuffier, brought to life a generation of surgeons which had hitherto been only a piece of history to us. Yet his questioning of Sir Stanford Cade on the rebuilding plans of the College showed that he had a knowledge of it much greater than many Fellows living within a bus ride of Lincoln's Inn. He recalled with pleasure and accuracy his discussions on hernia with Harold Edwards in war-time Rome. His delight at meeting Richard Handley brought back to both memories of a visit to Sampson Handley in

London when Richard Handley was a schoolboy. The conversation ranged over a wide field besides surgery. He talked of art and pictures, history and music, and he and his wife recounted anecdotes of Bernhard Berenson, the great authority on Florentine art, or told stories of the wiles of less reputable art dealers. He showed his guests his house, with all its treasures; but what perhaps made the deepest impression on us was his study, because in it lay the explanation of his modernity. It was a large room lined with books and its centre was occupied by a table covered with

Fig. 2. Professor Bastianelli with Lord Moynihan in 1916.

surgical journals from every part of the world, in sufficient confusion to show that they were closely read. Here, to illustrate a remark about Lord Moynihan, he picked up a photograph of Moynihan and himself (Fig. 2), garbed in operating gowns and dated 1916, and he unerringly pulled out books when he wished to make a point or clarify an explanation.

Professor and Signora Bastianelli are indeed remarkable and delightful people. The College has reason to be proud of its senior Honorary Fellow.

S.C., H.E. and R.H.

THE LATE SIR GORDON GORDON-TAYLOR

An appreciation from the Royal Australasian College of Surgeons

THE ASSOCIATION OF Sir Gordon Gordon-Taylor with the young surgeons of New Zealand and Australia began in the early years of this century, when he was a member of the Out-patient Surgical Staff of the Middlesex Hospital. The close of the First World War found many young medical officers in England, and they seized the opportunities offered, and took the F.R.C.S. And Gordon-Taylor was one of those who seemed to delight in helping these young " Colonials " to achieve this surgical goal. Thereafter, these young Fellows kept touch with him, so that when the Royal Australasian College was founded in 1927 he was well known to many Fellows of this College.

His first official association with this College was his visit in 1934, when he and Professor Buckmaster conducted a Primary F.R.C.S. examination in Melbourne and Dunedin. In all, he made five official visits to Australasia. And at all times gave this College the benefit of his experience, and his enthusiastic support. Valuable though these visits were, his impact upon the College was the result of his own lovable personality. Himself the intellectual product of the best type of Scottish up-bringing and education, he really loved to teach others, and to help the beginner. And just why he should have derived so much pleasure from teaching, and helping, young men of Australasia, we shall never know. He was almost fiercely proud of the tradition and reputation of the English College, and perhaps his heart warmed towards the young overseas men who aspired to the Fellowship of his College. Perhaps he just thought them to be worth helping. We shall never know, we can only give thanks that it was so. He had that wonderful gift of being able to take interest, genuine interest, in the problems of young post-graduate surgeons, and those not so young. He gave us the benefit of his advice and experience; he gave us many hours of pleasure in his company. And we gladly, and sadly, acknowledge our debt.

In 1949, six Fellows of this College, who had won the Hallett Prize, gave a sum of money to found a similar Prize for the Primary F.R.A.C.S., and asked the Council that it should be known as the Gordon-Taylor Prize. And during the last year, and largely at the instigation of the younger Fellows of this College, a portrait of Gordon-Taylor was painted by James Gunn, and was presented to the College at the Annual Meeting in August 1960. And he lived long enough to be informed of the enthusiastic reception of the portrait. We have recently learnt that Sir Gordon Gordon-Taylor bequeathed to this College the portrait of his wife, painted by Cadogan Cowper in 1922.

THE LATE SIR GORDON GORDON-TAYLOR

At the last Meeting of the Council of the College, held on 20th October 1960, the following resolution was carried:—

> " The Council of the Royal Australasian College of Surgeons has received the news of the death of Sir Gordon Gordon-Taylor with the greatest regret; and places on record its appreciation of, and gratitude for, his many kind services to the Fellows and Council of this College."

This brief minute records the termination of his physical association with this College. But the portraits and the prize are lasting ties which will keep fresh the memory of one who was regarded with the greatest respect and affection by the Fellows of this College.

PROCEEDINGS OF THE COUNCIL IN DECEMBER

AN ORDINARY MEETING of the Council was held on 8th December 1960, with Sir Arthur Porritt, President, in the Chair.

The first business was the admission of Lord Kindersley, Chairman of the Appeal Committee, to the Court of Patrons, the citation in his honour being given by Sir James Paterson Ross, Bt., Past President.

Two prizes were then presented—the Hallett Prize to Dr. J. L. Mercer of St. Thomas's Hospital Medical School and the Begley Prize to Dr. Joseph Brown of King's College Hospital Medical School.

The Council were informed of the award of the Lister Medal for 1960 to Professor Wilder Penfield of Montreal, an Honorary Fellow of the College.

Professor Digby Chamberlain of Leeds, Vice-President, was appointed Bradshaw Lecturer for 1961.

Sir Stanford Cade, Vice-President, was again nominated as examiner in surgery for the Fellowship of the Faculty of Radiologists.

Mr. G. T. Hankey, O.B.E., T.D., Fellow in Dental Surgery, was awarded the John Tomes Prize for 1957–59 in recognition of his clinical research, and Professor A. I. Darling, also a Fellow in Dental Surgery, was appointed Charles Tomes Lecturer for 1961. The Evelyn Sprawson Prize was awarded to Mr. M. L. Maberley of Guy's Hospital Dental School on the result of the recent Final Examination for the Licence in Dental Surgery.

THE LATE SIR GORDON GORDON-TAYLOR

An appreciation from the Royal Australasian College of Surgeons

THE ASSOCIATION OF Sir Gordon Gordon-Taylor with the young surgeons of New Zealand and Australia began in the early years of this century, when he was a member of the Out-patient Surgical Staff of the Middlesex Hospital. The close of the First World War found many young medical officers in England, and they seized the opportunities offered, and took the F.R.C.S. And Gordon-Taylor was one of those who seemed to delight in helping these young " Colonials " to achieve this surgical goal. Thereafter, these young Fellows kept touch with him, so that when the Royal Australasian College was founded in 1927 he was well known to many Fellows of this College.

His first official association with this College was his visit in 1934, when he and Professor Buckmaster conducted a Primary F.R.C.S. examination in Melbourne and Dunedin. In all, he made five official visits to Australasia. And at all times gave this College the benefit of his experience, and his enthusiastic support. Valuable though these visits were, his impact upon the College was the result of his own lovable personality. Himself the intellectual product of the best type of Scottish up-bringing and education, he really loved to teach others, and to help the beginner. And just why he should have derived so much pleasure from teaching, and helping, young men of Australasia, we shall never know. He was almost fiercely proud of the tradition and reputation of the English College, and perhaps his heart warmed towards the young overseas men who aspired to the Fellowship of his College. Perhaps he just thought them to be worth helping. We shall never know, we can only give thanks that it was so. He had that wonderful gift of being able to take interest, genuine interest, in the problems of young post-graduate surgeons, and those not so young. He gave us the benefit of his advice and experience; he gave us many hours of pleasure in his company. And we gladly, and sadly, acknowledge our debt.

In 1949, six Fellows of this College, who had won the Hallett Prize, gave a sum of money to found a similar Prize for the Primary F.R.A.C.S., and asked the Council that it should be known as the Gordon-Taylor Prize. And during the last year, and largely at the instigation of the younger Fellows of this College, a portrait of Gordon-Taylor was painted by James Gunn, and was presented to the College at the Annual Meeting in August 1960. And he lived long enough to be informed of the enthusiastic reception of the portrait. We have recently learnt that Sir Gordon Gordon-Taylor bequeathed to this College the portrait of his wife, painted by Cadogan Cowper in 1922.

THE LATE SIR GORDON GORDON-TAYLOR

At the last Meeting of the Council of the College, held on 20th October 1960, the following resolution was carried:—

> " The Council of the Royal Australasian College of Surgeons has received the news of the death of Sir Gordon Gordon-Taylor with the greatest regret; and places on record its appreciation of, and gratitude for, his many kind services to the Fellows and Council of this College."

This brief minute records the termination of his physical association with this College. But the portraits and the prize are lasting ties which will keep fresh the memory of one who was regarded with the greatest respect and affection by the Fellows of this College.

PROCEEDINGS OF THE COUNCIL IN DECEMBER

AN ORDINARY MEETING of the Council was held on 8th December 1960, with Sir Arthur Porritt, President, in the Chair.

The first business was the admission of Lord Kindersley, Chairman of the Appeal Committee, to the Court of Patrons, the citation in his honour being given by Sir James Paterson Ross, Bt., Past President.

Two prizes were then presented—the Hallett Prize to Dr. J. L. Mercer of St. Thomas's Hospital Medical School and the Begley Prize to Dr. Joseph Brown of King's College Hospital Medical School.

The Council were informed of the award of the Lister Medal for 1960 to Professor Wilder Penfield of Montreal, an Honorary Fellow of the College.

Professor Digby Chamberlain of Leeds, Vice-President, was appointed Bradshaw Lecturer for 1961.

Sir Stanford Cade, Vice-President, was again nominated as examiner in surgery for the Fellowship of the Faculty of Radiologists.

Mr. G. T. Hankey, O.B.E., T.D., Fellow in Dental Surgery, was awarded the John Tomes Prize for 1957–59 in recognition of his clinical research, and Professor A. I. Darling, also a Fellow in Dental Surgery, was appointed Charles Tomes Lecturer for 1961. The Evelyn Sprawson Prize was awarded to Mr. M. L. Maberley of Guy's Hospital Dental School on the result of the recent Final Examination for the Licence in Dental Surgery.

Diplomas of Fellowship were granted to 122 candidates. At the November examination six candidates out of 17 were successful in Ophthalmology, four out of 16 in Otolaryngology and 120 out of 409 in General Surgery.

There was one election to the Fellowship in Dental Surgery, Mr. I. R. H. Kramer of the Institute of Dental Surgery.

It was agreed that the new regulations for the Primary Examination for the Fellowship in Dental Surgery should come into force in September 1961.

The Council heard with great regret of the death of Dr. Louis Bazy, Honorary Fellow, a past President of the Académie de Chirurgie, and of Sir Harold Boldero, the Registrar of the Royal College of Physicians.

The following hospitals were recognized under paragraph 23 of the Fellowship regulations:

Hospitals	Posts Recognized		
	General (6 months unless otherwise stated)	Casualty (all 6 months)	Unspecified (all 6 months)
Derby—Derbyshire Royal Infirmary (additional)			2 Registrars (Neurosurgery)
London—Whipps Cross Hospital (additional)			*Under para.* 23 (*b*) Regr. (12 months) S.H.O. (6 months)
Halifax—The Royal Infirmary	1 Surg. Reg.		1 Orth. Reg. (additional)
Halifax—General Hospital (rearrangement of duties)	1 Surg. Reg.		

After the meeting, the Robert Jones Lecture was delivered by Mr. H. J. Seddon, C.M.G., on "The Manchester Ship Canal and the Colonial Frontier".

IMPERIAL CANCER RESEARCH FUND

On Sunday, 20th November 1960, an appeal was made in the B.B.C.'s "Week's Good Cause" programme on behalf of the Imperial Cancer Research Fund by Sir Cecil Wakeley, Bt., Chairman of the Council of the Fund. Sir Cecil spoke as follows:

"It is my privilege to speak to you as Chairman of the Council of the Imperial Cancer Research Fund and I speak as a surgeon with forty-five years' experience of the cancer problem.

"I seek your help in an urgent task.

"The Fund was established by the Royal College of Physicians of London and the Royal College of Surgeons of England in 1902. Before 1900 cancer had been widely investigated, but opinions regarding the nature and treatment of the disease were so conflicting that the mystery of its cause and cure seemed insoluble. The Colleges realized that systematic research was vital, and the Fund was created to undertake this work.

" It was soon apparent that cancer presented a problem of unimagined complexity. Each adventure into a likely field of research opened up vast new fields to be explored. The pathologist and virologist were joined by the biochemist and biophysicist. Indeed, every branch of medical science was to be called upon, and the work to-day demands co-ordinated scientific and medical skill at the most advanced level.

" The results of such research are rarely spectacular until the final goal is reached; but a tremendous amount of knowledge has been accumulated, and through it thousands of lives have been saved or prolonged, while the sufferings of countless patients have been alleviated.

" The Fund's work, which is carried on in co-operation with the great teaching hospitals, is directed by Dr. Guy Marrian, a Fellow of the Royal Society. Under Dr. Marrian are the Heads of Divisions, each with a staff of scientists, doctors and technicians. Within the Divisions particular forms of cancer are under continuous investigation. For example, there are specialists dealing with lung cancer, others with cancers of the breast, others with leukaemia which takes such a tragic toll of young lives.

Sir Cecil Wakeley broadcasting his appeal on behalf of the Imperial Cancer Research Fund.

" All of this work is to be greatly expanded. We are building laboratories which will be the most advanced cancer research centre in Europe, and this expansion will enable us to double the number of our research staff. The laboratories and equipment will cost a million and a quarter pounds to build, and, when they are ready, our annual expenditure, now two hundred thousand pounds, will be more than doubled.

" The Fund relies entirely upon the public for support and I want you to know that our administrative costs, including advertising and appeals, represent only 6 per cent. of income. Your money is spent on research.

" A million and a quarter pounds! Annual expenditure to be doubled! You see why we need a very great deal of money. But is any sum too great if it will help to rid mankind of a scourge which brings sufferings and sorrow to the homes of people everywhere?

" The work of the Fund has cleared away confusions and ignorances. It has brought knowledge—it has brought a great measure of healing—supremely, it has brought hope. We know now the direction in which we are travelling, and we know that at the end of the road is the goal we all so earnestly desire. But we do not yet know how far we have to travel. What we can do—and what we must to—is to speed our journey, and your help will enable us to do this. Please send your gifts to me, Sir Cecil Wakeley, Imperial Cancer Research Fund, Lincoln's Inn Fields, London."

ANNUAL MEETING OF FELLOWS AND MEMBERS

THE ANNUAL MEETING of the College was held on Wednesday, 7th December, at the College. With the exception of the Department of Physiology, which was closed owing to rebuilding operations, the Scientific Departments were on view all day, and visitors were able to see the research work being carried on at the College. There was an exhibition of historical books and manuscripts in the Library.

Two lectures forming part of the Basic Medical Sciences Course were open to visitors: the first of these was on "The arm and forearm", by Professor R. J. Last, and the second on "Hormones, sex hormones and insulin", by Dr. K. Hellman.

A large audience gathered in the Edward Lumley Hall in the afternoon to hear Sir Stanford Cade, K.B.E., C.B., F.R.C.S., Vice-President, deliver the Bradshaw Lecture. Sir Stanford chose as the title of his lecture "Malignant melanoma", and it is hoped to publish this lecture in a future issue of the *Annals*.

The Annual Meeting of Fellows and Members took place at 3.30 p.m., but unfortunately there were not enough people present to form a quorum. Sir Harry Platt, Bt., Past President, presented a report on the work of the International Federation of Surgical Colleges, and the Librarian, Mr. W. R. Le Fanu, spoke on the work of the Library in connection with the teaching and research being carried out in the College. The Chairman of the Finance Committee, Mr. C. Naunton Morgan, then spoke about the present financial position of the College and reported on the progress of the Appeal.

The Monthly Dinner of the College was held in the Edward Lumley Hall in the evening, and was very well attended. After the dinner, Dr. John Hunt, co-opted member of Council representing General Practice, gave a most fascinating talk on his recent visit to Russia, and this was illustrated with colour slides taken by Dr. Hunt during his visit.

There was an exhibition on view in Committee Room II of books, pictures and other items which have been presented to the College by some Russian colleagues and by the Academy of Science in Moscow. Of special interest were a case of surgical instruments for operations on the cardia and oesophagus, presented by Professor Andrey Savinych of Tomsk while on a visit to the College in April, 1956, and various items connected with Charles Darwin presented by Professor Alexander Kohts, the Curator of the Darwin Museum in Moscow.

GRANT OF FELLOWSHIP DIPLOMAS

AT THE FINAL EXAMINATION for the Fellowship held in November, six candidates out of seventeen were successful in Ophthalmology, four candidates out of sixteen in Otolaryngology and 120 out of 409 in General Surgery.

At the meeting of Council on 8th December, Diplomas of Fellowship were granted to the following:

*RUBEN, Cecil Montague (*Guy's*).
†STEEL, Anthony Edgar (*St. Mary's*).
BATSTONE, John Harold Frank (*St. Mary's*).
†BOSHER, Stephen Keith (*Guy's*).
GARDNER, Donald Cecil (*King's College*).
JAMES, Roger Alan (*Sheffield*).
†WALDECK, Henry John Smith (*Birmingham*).
JEFFREYS, John Goldsmith (*King's College*).
MEE, William Michael (*St. Mary's*).
DENMAN, Eric Edward (*University College*).
BAKER, Geoffrey Cecil Winchester (*Guy's*).
ANDREWS, Brian George (*Westminster*).
ROBERTS, Gwynn Richard Ellis (*The London*).
WAKELY, Molly Elizabeth (*University College*).
LETTIN, Alan William Frederick (*University College*).
MBANEFO, Theodore Chukwuemeka Ezeokpue (*Leeds*).
GULATI, Ajeet-Bans Lall (*Charing Cross*).
WASTELL, Christopher (*Guy's*).
CORT, Alexander (*Witwatersrand*).
ORR, Gillespie Neil McGilp (*Sydney*).
RAMAIYA, Samuel Gopal (*Madras*).
DE BRUIN, Arthur James (*Ceylon*).
LIVINGSTONE, Peter Deans (*Edinburgh*).
SAMANTA, Birinchi Prasad (*Calcutta*).
KRAMER, Hyman (*Witwatersrand*).
ADAMS, David James (*Liverpool*).
BATES, Arthur (*St. Bartholomew's*).
MACLAREN, Iain Ferguson (*Edinburgh*).
McRAE, Ronald Kenneth (*Glasgow*).
PURI, Harish Chandra (*Lucknow*).
SUMARIWALLA, Dhun Dinshaw (*Bombay*).
CHESTERMAN, Patrick James (*St. Mary's*).
KAZIM, Ahmed Abdulla (*Bombay*).
MASTERTON, John Potter (*Glasgow*).
*FENTON, Colin Robert (*New Zealand*).
HAGGART, Brian Gerard (*Liverpool*).
HAW, David William Martin (*Leeds*).
HO, Hon Fah (*Malaya*).
MANDER, Jeffory George (*St. Mary's*).
SHANMUGALINGAM, Thamotherampillai Nadarajah (*Ceylon*).
SUSSMAN, Trevor (*Westminster*).
BRADLEY, John William Paulton (*Westminster*).
DINNING, Graham Gordon (*Sydney*).
LU, Earl Ming-Teh (*Sydney*)
*CROCK, Gerard William (*Melbourne*).
CROSBY, David Lewis (*Wales*).
FOSTER, David Dryhurst (*Birmingham*).
GOONEWARDENE, Roy Welborn (*Ceylon*).
GRABHAM, Anthony Herbert (*Durham*).
HAGUE, John Malcolm Seaforth (*Middlesex*).
HEYWOOD-WADDINGTON, Michael Broke (*Middlesex*)

HOWE, Colin Thomson (*Middlesex*).
HUNT, John Aspinall (*Witwatersrand*).
KIRWAN, Ernest O'Gorman (*Middlesex*).
MODY, Adi Eruch (*Bombay*).
MULLINS, Allan Edwin Joseph (*Sydney*).
REDDY, Sannapareddi Sreenivasulu (*Andhra*).
RENTON, Charles James Crawford (*Glasgow*).
SCHRAIBMAN, Ivor Gerald (*Witwatersrand*).
SIVARAMAN, Letchumanan (*Madras*).
BETTY, Michael John (*Bristol*).
*BOUGHER, Gordon James (*Melbourne*).
†CAUST, Leslie James (*Adelaide*).
COHNEY, Benjamin Coplan (*Queensland*).
DECKER, Gustav Adolf George (*Cape Town*).
DICKINSON, Kenneth Montague (*Manchester*).
GIBBONS, John Robert Pelham (*Leeds*).
HAY, Donald Urquhart (*New Zealand*).
HOSSACK, Donald William (*Melbourne*).
LAINE, John Bunch (*Liverpool*).
*McAULIFFE, David Jeremiah (*Adelaide*).
*OWEN, Robin Arthur (*King's College*).
PAI, Ballambetto Yogish (*Madras*).
POIRIER, Henry (*St. Bartholomew's*).
RITCHIE, Douglas Malcolm (*Melbourne*).
THOMS, George Ronald (*Melbourne*).
ANNETTS, David Lyle (*Sydney*).
BAHL, Baldev Raj (*Calcutta*).
BINNS, Philip Metcalfe (*Leeds*).
COLLINS, Frederick John (*Sydney*).
DUKE, Stanley Rowland (*Queensland*).
EL-SHERIF, Hassanein Mohamed Hassanein (*Cairo*).
FENNELL, William Michael Pitt (*Cape Town*).
GLENN, David Campbell (*Sydney*).
GREEN, Mathew Christopher (*Melbourne*).
HARRIS, Walter Graham (*St. Bartholomew's*).
HIGGINS, Peter McRorie (*The London*).
HUDSON, Christopher Neville (*St. Bartholomew's*).
KILLE, John Narkett (*Birmingham*).
LENAGHAN, Richard (*Melbourne*).
LOGAN, Charles James Hume (*Belfast*).
LYNCH, Gearoid (*Dublin*).
McKIBBIN, Brian (*Leeds*).
MADHOK, Prithvinath Dinanath (*Bombay*).
MAGEE, Harold Reginald (*Queensland*).
MEFFAN, Peter Mann (*New Zealand*).
ORMANDY, Gordon James (*Sydney*).
REID, William Henry (*Glasgow*).
ROTHWELL-JACKSON, Richard Loxton (*St. Bartholomew's*).
ROWELL, Charles Edward (*Sydney*).
ROZNER, Leo (*Melbourne*).
SCOTT, Peter Milton (*Charing Cross*).
SHAH, Bhanu Ratilal (*Gujarat*).
SMITH, Harry Lawrence McIntyre (*New Zealand*).
WYATT, Arthur Powell (*St. Bartholomew's*).
AHMED, Hameed Ud-Deen (*Gauhati*).
CANAGARETNA, Christopher Wilmot Packiaratnam (*Ceylon*).
CHAUDHRY, Jagdish Kumar (*Lucknow*).
CHU, Eugene Poh Hwye (*Melbourne*).
JOHARI, Sohan Lal (*Rajputana*).
KING, Kevin Francis (*Melbourne*).
LI, Franklin Wang-Pong (*Hong Kong*).
PEASE, (The Hon.) William Simon (*St. Thomas's*).

GRANT OF FELLOWSHIP DIPLOMAS

ROGERS, Noel Michael Adam (*Witwatersrand*).
RUSSELL, John Alfred (*The London*).
SOUTHWOOD, Richard Taunton (*Adelaide*).
TARAPORVALA, Jamsheed Cooverji (*Bombay*).
VALIATHAN, Marthanda Varma Sankaran (*Travancore*).
VENTER, Pieter Jacobus (*Pretoria*).
SMITH, Ivo (*St. Mary's*).
STRANC, Miroslaw Felix (*Dublin*).
TALWALKER, Vasant Charuchandra (*Bombay*).

*In Ophthalmology. †In Otolaryngology.

RESPONSE TO THE BROADCAST BY SIR CECIL WAKELEY, Bt.

THE POPULARITY OF television has meant a very considerable decrease in the number of those who listen to sound broadcasts, and this has been most clearly reflected in the results of " Week's Good Cause " Appeals which come at a time when both B.B.C. and I.T.V. expect peak viewing for their popular television programmes. A review of the National (i.e. All Home Stations) " Week's Good Cause " Appeals since January 1959 shows that, apart from two exceptional appeals, the St. Martin-in-the-Fields Christmas Fund, £22,693, and Lady Churchill's appeal for World Refugee Year, £59,079, only one of the other sixty-six broadcasts achieved more than £8,000. Gifts are still being received in response to Sir Cecil's broadcast and the figure stood at £23,043 1s. 1d. on 23rd December 1960.

BOOKS ADDED TO THE LIBRARY

July—December 1960

Anaesthesia

BALLANTINE AND JACKSON. A practice of general anaesthesia for neurosurgery. Gift of Prof. R. F. Woolmer.
BOURNE. Nitrous oxide in dentistry.
BOUVET. Etat actuel du problème du curare.
OSTLERE. Anaesthetics. 4th edition.

Anatomy

CURUTCHET. Simplified anatomic instrumental. (La Plata, Argentina.) Author's gift.
ELLIS. Clinical anatomy.

Biochemistry

GOODWIN. Recent advances in biochemistry. 4th edition.
KOREY. Biology of myelin.
THORPE. Biochemistry. 7th edition.

Cancer

HADDOW. Chemical mechanisms in the induction of cancer. (Frankland lecture.)
INSTITUT GUSTAVE-ROUSSY. Traitement des Cancers. Gift of the French Embassy.
NOWINSKI. Fundamental aspects of normal and malignant growth.
RAVEN. Cancer: Progress volume. Editor's gift.
SUTHERLAND. Cancer, the significance of delay. Gift of the publishers, Messrs. Butterworth.

Cytology

BRACHET AND MIRSKY (editors). The Cell, vol. 4 (continuation of series).
WILLMER. Cytology and evolution.

BOOKS ADDED TO THE LIBRARY

Dental Research

JAMES. The jaws and teeth of primates.

JENKINS. Physiology of the mouth. 2nd edition.

Gastroenterology

AVERY JONES AND GUMMER. Clinical gastroenterology.

Gynaecology

SHAW'S Textbook of operative gynaecology. 2nd edition, by Howkins.

Histology

BOURNE. Introduction to functional histology. 2nd edition.

Hypothermia

COOPER AND ROSS. Hypothermia in surgical practice.

STARKOV (editor). Problem of acute hypothermia. Translated from Russian.

Infection

WILLIAMS AND OTHERS. Hospital infection.

Medical Illustration

MCLARTY. Illustrating medicine and surgery. Gift of Sir Cecil Wakeley, Bt.

Neurology

BONICA. Clinical applications of diagnostic and therapeutic nerve blocks. 1958. Gift of Prof. Lambert Rogers.

CHUSID AND MCDONALD. Correlative neuro-anatomy. 10th edition.

SEMMES. Somato-sensory changes after penetrating head wounds. Gift of the London School of Hygiene.

WYKE. Principles of general neurophysiology.

ZANGWILL. Cerebral dominance and physiological function.

Ophthalmology

ANDERSON. Ocular vertical deviations. 2nd edition.

SORSBY AND UNGAR. Antibiotics and sulphonamides in ophthalmology Gift of Prof. Arnold Sorsby.

Orthopaedics

AMERICAN COLLEGE OF SURGEONS. An outline of the treatment of fractures. Gift of the A.C.S.

MEURIG WILLIAMS. The treatment of traumatic paraplegia due to fracture dislocation of the spine. (Illustrated typescript thesis.) Author's gift.

Pathology

CIBA. Symposium on congenital malformations.

HARRISON. Recent advances in pathology. 7th edition.

Pharmacology

British Pharmacopoeia, Addendum 1960.

Physiology

BOURNE. Structure and function of muscle, vols. 2–3 (completion of series).

MCDONALD. Blood flow in arteries.

MARSHALL. Physiology of reproduction, 3rd edition, by Parkes, vol. 1, part 2 (continuation).

PITTS. Physiological basis of diuretic therapy.

RUCH AND FULTON. Medical physiology (Howell's Physiology, 18th edition).

Radiology

MCLAREN. Modern trends in diagnostic radiology. 3rd series.

Research Methods

ATKINS (editor). Tools of biological research. 2nd series. Gift of the editor, Mr. H. J. B. Atkins.

Surgery

ALEXANDER AND NORMAN. The Sturge-Weber syndrome. Gift of Messrs. John Wright, publishers, and of Sir Cecil Wakeley, Bt.

AMERICAN COLLEGE OF SURGEONS. Early care of acute soft tissue injuries, 2nd edition. Gift of A.C.S.

BAILEY. Demonstrations of physical signs in clinical surgery. 13th edition.

BROWN AND FRYER. Post-mortem homografts.

HARLEY. Modern trends in cardiac surgery.

HUDEMANN. Chirurgische Nahtmaterial.

ROB AND SMITH. Operative Surgery: service volume. Gift of Mr. M. L. Formby (continuation).

BOOKS ADDED TO THE LIBRARY

Urology

RICHES (editor). Modern trends in urology, 2nd series. Gift of Sir Eric Riches.

Zoology

OSMAN HILL. Primates, vol. 4: Cebidae (continuation).

Reference Books

AMERICAN COLLEGE OF SURGEONS. Catalogue of the H. Winnett Orr Historical collection and other rare books in the library, by L. M. Prime. Gift of the A.C.S.

FRENCH'S Index of differential diagnosis, 8th edition, by Douthwaite.

Hospital Gazetteer (B.M.A.).

National Library of Medicine, Washington. Cumulative Catalog. 1955–59. 6 vols.

Biography and Bibliography

HOLTHUSEN. Ehrenbuch der Röntgenologen aller Nationen. 2nd edition.

HERMANN BOERHAAVE: Bibliographia Boerhaaviana, by G. A. Lindeboom.

S. G. BROWNE, F.R.C.S.: Duncan. Bonganga, experiences of a missionary doctor. Gift of Dr. Browne.

R. W. COLE, F.R.C.S. Kossoh-town Boy (autobiography).

A. VON HALLER: Verzeichnis der gedruckten Schriften Albrecht von Hallers, by S. Lundsgaard-Hansen.

ROBERT HOOKE: A Bibliography of Dr. Robert Hooke, by Sir Geoffrey Keynes. Author's gift.

JOHN HUNTER: The reluctant surgeon, by John Kobler.

RENÉ LERICHE: Leriche chirurgien, by H. Mondor.

ST.GEORGE MIVART: A conscience in conflict, by J. W. Gruber. Author's gift.

D. W. G. MURRAY, F.R.C.S. Medicine in the making (autobiography). Author's gift.

JOSEPH PRIESTLEY: The crucible, the story of Joseph Priestley, F.R.S., by H. Gillam.

JAN EVANGELIST PURKINJE: J. E. Purkyne, his life and works, by H. J. John.

History of Medicine and Science

ANDRADE. A brief history of the Royal Society.

BISHOP. The early history of surgery.

COPEMAN. Doctors and disease in Tudor times.

FAXON. The Massachusetts General Hospital 1935–55. Presented by the Hospital in exchange for Sir Zachary Cope's History of the College.

FLORKIN. Naissance et déviation de la théorie cellulaire dans l'oeuvre de Théodore Schwann.

MORSON. St. Peter's Hospital.

ROTHSCHUH. Geschichte der Physiologie.

SYKES. Essays on the first hundred years of anaesthesia. Gift of Prof. Digby Chamberlain.

HOWARD GRAY LIBRARY

Authors and Writers Who's Who 1960.

CHAMPION. Racial proverbs. 1950.

BURY. The Zenith of European power. (New Cambridge Modern History, vol. 10.)

WATSON. The reign of George III. (Oxford History of England, vol. 12.)

DOWN HOUSE LIBRARY

DARWIN. Letters to Professor Henslow (Reprint of pamphlet of 1835). Gift of the Cambridge Philosophical Society.

DARWIN. Notebooks, parts 3 and 4, edited by Sir Gavin de Beer (continuation). Gift of the British Museum (Natural History).

DONATIONS

DURING THE LAST few weeks the following generous donations have been received:

Appeal Fund—Donations:

£5,000	Miriam Sacher Charitable Trust (further gift)
£2,000	Arawak Trust Co., Ltd.
£1,450	Joseph Collier Trust (further gift)
£1,000	Lloyds Charitable Trust (further gift)
£250	The Fishmongers' Company Phoenix Assurance Co., Ltd. (further gift) Power Securities, Ltd.
£105	West Riding Worsted & Woollen Mills, Ltd. (further gift) W. H. Foster, Esq. Associated Engineering, Ltd. E. W. Bryan, Ltd.
£100	Steel & Busks, Ltd. Associated British Machine Tool Makers, Ltd. Harold J. Cotes, Esq. Transport Development Group, Ltd. The Mond Nickel Co., Ltd. (further gift)
£52 10s. 0d.	Walsall Conduits, Ltd.
£50	Miss Agnes Little. International Bottle Co., Ltd. Crofts (Engineers), Ltd. Hopkinsons, Ltd.
£41	Cornhill Insurance Co., Ltd.
£26 5s. 0d.	Killick Martin & Co., Ltd. (further gift) White, Tomkins & Courage, Ltd. H. Harrison & Co. (Finishers), Ltd. G. Durston & Son, Ltd. John Lewis Partnership, Ltd.
£25	Castles Motor Co. (Leicester), Ltd. Pick, Everard, Reay & Gimson Howe Robinson & Co., Ltd. Cotman Investment Trust, Ltd.
£21	S. A. Squirrell & Co., Ltd. Mrs. F. U. Francis.
£20	D. Hendersons & Sons, Ltd. Mars, Ltd.
£15 15s. 0d.	G. & R. Dewhurst, Ltd.
£10 10s. 0d.	Thomas Ramsden & Son, Ltd. Macready's Metal Co., Ltd. Bennett Bros. (Hosiery), Ltd. Foster Wheeler, Ltd. George G. Harrap & Co., Ltd. Charles Letts & Co., Ltd. Imperial Life Assurance Company of Canada Foy, Morgan & Co., Ltd. G. W. Owen, Esq.
£10	Express & Star Charitable Trust The Broderers Company
£6 2s. 6d.	The Gotham Co., Ltd. (further gift)

£5 5s. 0d.	William Oliver & Sons, Ltd. The Guilford Stores, Ltd. Harris, Ltd. A. Weston Evans, Esq. Argee Co., Ltd. James Lulham & Co., Ltd. Howards (Colney), Ltd. Millers Timber Trading Co., Ltd. Bradley & Co., Ltd. Hunter-Penrose, Ltd. Sneyd Brickworks, Ltd. Percy Jones (Twinlock), Ltd. W. H. Fraser, Esq. W. F. Hall, Esq. (further gift)
£5	Sheffield Photo Co., Ltd. Colonel J. Dalton White
£4	Spennymoor Women's Institute
£3 3s. 0d.	H. Samuel, Ltd. (Birmingham)
£2 2s. 0d.	Frederick Lawrence, Ltd. W. Williams & Sons, Ltd. Simpkin, Machin & Co., Ltd.
£1 1s. 0d.	Sunlight Launderies (Lond. & Western), Ltd.
£1	Mrs. G. H. Wardle
10s. 0d.	H. C. Beaumont, Esq. (further gift)
7s. 6d.	Miss R. Thompson

Appeal Fund—Covenants:

£80 p.a. for 7 years + tax	The British Ratin Co., Ltd.
£50 p.a. for 7 years + tax	Standard Fireworks, Ltd. The British Combined Investors Trust, Ltd. Air Products (Great Britain), Ltd.
£25 p.a. for 7 years + tax	The Anglo-Celtic Trust, Ltd. Rio Claro Investment Trust, Ltd.
£21 p.a. for 7 years + tax	G. Stibbe & Co., Ltd.
£20 p.a. for 7 years + tax	Romney Trust, Ltd.
£10 10s. 0d. p.a. for 7 years	R. N. Hill, Esq.
£10 p.a. for 7 years + tax	J. H. Caramel, Esq.
£10 p.a. for 7 years	H. & J. Quick, Ltd.
£3 3s. 0d. for 7 years + tax	Lloyd Attree & Smith, Ltd.

McIndoe Memorial Fund

£5	A. H. Hunt, Esq., F.R.C.S.

Voluntary donations or subscriptions under Covenant by Fellows:

The following have generously given a donation to the College or have undertaken an annual subscription under covenant:

H. N. Bloom, F.F.A.R.C.S. R. Barnes, F.R.C.S.

DIARY FOR JANUARY

Tues.	17		Final F.F.A. Examination begins.
		5.00	Dr. D. C. Caldwell—Erasmus Wilson Demonstration—Radio-active isotopes in a chemical laboratory.
Fri.	20	5.00	Board of Faculty of Dental Surgery.
Mon.	23		Basic Sciences Lectures and Demonstrations begin.
Wed.	25		Primary F.R.C.S. Examination begins.
		5.00	Professor G. W. Causey—Arnott Demonstration—An appreciation of the fine structure of some of John Hunter's specimens.
Thur.	26	5.00	Mr. N. G. Rothnie—Arris and Gale Lecture—Changes in blood coagulation due to perfusion for open heart surgery.

DIARY FOR FEBRUARY

Wed.	1		Final L.D.S. Examination (Part I) and D.O. Examination begin.
Thur.	2	5.30	Professor Victor Lambert—Otolaryngology Lecture—Malignant melanoma of the nose.
Fri.	3		Basic Sciences Lectures and Demonstrations for Dental Students end.
Tues.	7	5.00	Dr. L. Bitensky—Erasmus Wilson Demonstration—Cirrhosis of the liver.
Wed.	8		Final L.D.S. Examination (Part II) begins.
Thur.	9		D. Phys. Med. Examination (Part I) begins.
		2.00	Ordinary Council.
		5.00	Mr. P. H. Golding-Wood—Arris and Gale Lecture—Anatomic surgery of the ear, nose and throat.
Mon.	13		Course in Clinical Surgery begins.
Tues.	14		Hunterian Festival: John Hunter born 1728.
		4.00	Hunterian Oration—Sir Russell Brock—Museum and Research.
Wed.	15		D.M.R.D. Examination (Part I), D.M.R.T. Examination (Part I) and D.T.M. & H. Examination begin.
Thur.	16		D. Phys. Med. Examination (Part II) begins.
Fri.	17	5.00	Dr. Hamilton B. G. Robinson—Webb-Johnson Lecture—The effect of operative procedures on the pulp.
Mon.	20	5.00	Professor Arnold Sorsby—Ophthalmology Lecture—Experimental degeneration of the retina and its clinical implications.
Tues.	21	5.00	Professor Arnold Sorsby—Ophthalmology Lecture—The nature of refractive errors: the present position.
Wed.	22		Primary F.D.S. Examination and D.M.R.D. Examination (Part II) begin.
Thur.	23	5.00	Professor C. E. Drew—Hunterian Lecture—Profound hypothermia in cardiac surgery.
Tues.	28	4.15	Dr. R. McP. Livingston—Arnott Demonstration—Conjoint twins.

"The only antibiotic that combats Pseudomonas infection really effectively is Polymyxin....

.... known as 'AEROSPORIN'.... available as Polymyxin B Sulphate powder in vials containing 500,000 units. It may be given by mouth and by intravenous, intramuscular, intrathecal, subconjunctival, and intrapleural routes.... may be given by mouth for bowel infections which do not respond to sulphonamides, streptomycin, or neomycin.....The indication for its use in severe infections that resist other antibiotics usually outweighs the risk of toxicity."

Annot., Lancet, i, 375, 1960.

'AEROSPORIN'

Polymyxin B Sulphate

BURROUGHS WELLCOME & CO. LONDON

(The Wellcome Foundation Ltd.)

For major electro-surgery

RADIOTOM 611

* 4 different types of current for cutting and coagulation
* Endoscopy up to 12v, 1 amp, continuously variable
* Safety circuit prevents accidental burns due to broken indifferent electrode cable
* Extra light and cautery circuits may be added
* Special model available for flush mounting in wall of operating theatre

Write or telephone for further details

SIEREX LTD

241 Tottenham Court Road
London W.1 LANgham 2464

THE MEDICAL DEFENCE UNION

The Oldest and Largest Organisation of its kind in the World

Secretary : Philip H. Addison, M.R.C.S., L.R.C.P.

Tavistock House South, Tavistock Square, W.C.1

Butterworth Books for Surgeons

PATHOLOGY OF TUMOURS

By Professor R. A. WILLIS, D.SC., M.D., F.R.C.P. *3rd edition.* 1056 *pages.* 500 *illustrations. Price* 105*s.*

Since its first appearance, this great work has been regarded in medical circles throughout the world as one of the most outstanding contributions to pathological literature in recent years. For this Third Edition, Professor Willis has thoroughly revised the text, taking in all the new advances and developments, and presenting once again a complete and most lucid picture of tumour pathology in the light of present-day experience and research.

CANCER PROGRESS 1960

By RONALD W. RAVEN, O.B.E.(MIL.), T.D., F.R.C.S. 273 *pages.* 45 *illustrations. Price* 60*s.*

CANCER PROGRESS 1960 is a report on the new advances in knowledge and international research in the cancer field. The construction of the volume is closely linked to *CANCER* (*Ed. R. W. Raven, 6 vols.+index—Butterworths* 1957–59) in that the material is grouped under the main headings: Experimental Research, Pathology, Statistical and Industrial Aspects, Clinical Aspects, Diagnosis, Radiotherapy, Chemotherapy and Social Aspects. It is in fact an extension of Raven's *CANCER*, but at the same time a new serial publication which will be purchased by many in the medical profession seeking up-to-date information on the contemporary cancer field.

CANCER—THE SIGNIFICANCE OF DELAY

By ROBERT SUTHERLAND, M.D., D.P.H. 222 *pages.* 59 *tables. Price* 30*s.*

Some recent evidence has been offered which indicates that the behaviour of cancer is " predetermined ", and that early diagnosis may not be all-important. Dr. Sutherland has probed the facts, including what is known about the interaction of host and tumour. His clearly written assessment of the reliability or weakness of the evidence provides an unusually penetrating and worthwhile contribution to the literature.

MODERN TRENDS IN CARDIAC SURGERY

Edited by H. R. S. HARLEY, M.S., F.R.C.S. 304 *pages.* 79 *illustrations. Price* 70*s.*

This is the first book on this subject to be produced in Great Britain, and the consultant, the trainee, and the general practitioner will find much in this account of British thought and practice to stimulate his interest in, and further his knowledge of, cardiac surgery. The subjects dealt with cover aspects of current interest, giving full attention to new techniques such as hypothermia, deep hypothermia and extra corporeal circulation.

Full details available, post free, from

BUTTERWORTHS · 4-5 BELL YARD, LONDON, W.C.2

THE BEST INVESTMENT I MADE

An Insurance Policy with my profession's own Insurance Society. *I've never regretted it.*

They specialise in

Non Cancellable With Profit
SICKNESS AND ACCIDENT INSURANCE.
LIFE ASSURANCE.
PERSONAL PENSION POLICIES.

When you are buying a car why not ask for details of the Hire Purchase Scheme of our subsidiary company — the Medical Sickness Finance Corporation, of 7 Cavendish Square, London, W.1. Telephone Museum 9348.

MEDICAL SICKNESS ANNUITY AND LIFE ASSURANCE SOCIETY LIMITED

Write for particulars, mentioning this advertisement

Telephone : Langham 0341

3 CAVENDISH SQUARE, LONDON, W.1.

Printed by Jackson, Ruston & Kossen, Ltd., Pear Tree Court, London, E.C.1, England.